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A CASE OF SPONTANEOUS LACTATION ASSOCIATED WITH MENSTRUAL IRREGULARITY AND STERILITY

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Spontaneous and persistent lactation not associated with pregnancy or acromegaly appears to be a relatively uncommon condition. We report a case where galactorrhoea was the presenting symptom of an obese nulliparous patient.

CASE REPORT

A 35-year-old married woman was admitted to hospital in January 1957 for investigation of a spontaneously occurring galactorrhoea. The patient was an obese Coloured woman who complained of fullness of the breasts and a milky discharge, especially from the left nipple. This condition had been present for 18 months and during this time the discharge had fluctuated in intensity, being most active in the premenstrual cycle. The secretion had ceased spontaneously for a week or two on several occasions, but during the past 3 months the discomfort had increased and the milky discharge become more profuse. Sterility and menstrual irregularity were prominent features of the history. Menstruation had commenced at the age of 11 years, lasted 6-7 days, and pursued a regular 30-day cycle. In recent years, however, the flow had diminished, the cycle become irregular, with frequent episodes of amenorrhoea, and 7 years of married life had failed to result in a pregnancy.

The patient was found to be an obese Coloured woman who weighed 209 lb. and measured 59½ inches in height. Clinically she exhibited none of the features of acromegaly, and on examination the possibility of pregnancy was excluded. Oiliness of the facial skin was associated with a mild form of seborrhoeic dermatitis. Striae and hirsutism were absent. The breasts were pendulous but of normal consistency, and yielded a milky secretion on gentle pressure. Although not profuse, pressure always produced this secretion, varying in amount from a few drops to a thin stream on each occasion. Pelvic examination combined with an endometrial biopsy was performed at a later date, under general anaesthesia, and no gross abnormality of the genital organs was detected. The blood pressure was 150/90 mm. Hg, and examination of the nervous system including fundoscopy and examination of the visual fields, revealed nothing abnormal.

The following investigations were carried out:

X-ray of chest and skull: No abnormality noted.

Coned views of pituitary fossa: No abnormality noted.

Urinalysis on repeated examination showed nothing of note.

Urinary 17-ketosteroids: 8·8 mg. in 24 hours and 5·2 mg. in 24 hours (estimated as dehydroisoandrosterone).

Urinary FSH: 6-12 mouse units in 24 hours (on 3 occasions).

Xenopus frog test: negative.

Full blood-count: Within normal limits.

Blood Urea: 27 mg. per 100 ml.

Serum sodium: 130 mEq. per litre.

Serum potassium: 4·6 mEq. per litre.

Serum chloride (as Na Cl): 100 mEq. per litre.

Serum protein—bound iodine: 6·6 μg. per 100 ml. and 3·7 μg. per 100 ml.

Serum cholesterol: 300 mg. per 100 ml.

Serum inorganic phosphorus: 3·2 mg. per 100 ml.

Serum calcium: 5·1 mEq. per litre.

Blood serological test: Negative modified Ide.

Blood sugar: 68 mg. per 100 ml. (fasting).

Glucose tolerance test: A normal curve.

Insulin sensitivity test: No evidence of 'hypoglycaemia unresponsiveness'.

Basal metabolic rate: +1.

Endometrial biopsy: This was performed on the 1st day of the menstrual cycle and the minute pieces of endometrium submitted for study showed nothing of significance.

DISCUSSION

A detailed account of current opinion regarding the complex endocrine control of mammary growth and lactation is beyond the scope of this report, but certain views call for mention. The development of the breast is controlled by oestrogen, which leads to growth and branching of the mammary duct system, while progesterone leads to glandular development. During pregnancy the placenta forms both oestrogen and progesterone, thus stimulating duct proliferation and the glandular cells. An anterior pituitary gonadotrophin, prolactin, is thought to be responsible for the stimulus to the alveolar epithelium to synthesize milk. Prolactin is probably formed by the eosinophil cells of the anterior pituitary gland, and acts only on breasts that have been previously conditioned by oestrogen-progesterone

stimulation, and is itself luteotrophic, stimulating the release of progesterone from the corpus luteum of the ovary. There is evidence that prolactin secretion is stimulated by suckling, probably *via* a nervous pathway to the hypothalamus and finally controlled by circulating blood-hormone levels. Recent work by Bensen *et al.* (1957) suggests that oxytocin, a posterior pituitary hormone stimulates the release of prolactin. Lactation does not occur simply as a function of prolactin but apparently requires the synergistic action of both prolactin and the adrenal cortex (Gomez and Turner, 1937, Nelson, and Gaunt, 1937). A complex relationship exists between the pituitary and the ovary and it is known that oestrogen has a profound influence upon pituitary activity. After castration there occurs a marked increase in the gonodotrophic activity of the pituitary, and Zondek (1932) observed an increase in the urinary excretion of FSH in both sexes after castration; at the menopause a similar increase is found. Nelson (1936) believes that the reason why lactation does not occur during the last months of pregnancy is the inhibiting effect of large quantities of oestrogen on the pituitary gonadotrophin, prolactin; the decrease in systemic oestrogen which follows parturition permits the pituitary to release prolactin and stimulate lactation. There is thus sufficient evidence to show that the gonadotrophic activity of the anterior pituitary is sensitive to the circulating ovarian hormones, especially oestrogen, and under certain conditions this balance may be disturbed.

Numerous cases of spontaneously occurring lactation, or alteration in mammary activity, have been recorded in both sexes. Mechanical stimulation of the nipple is cited as a common cause, and Knott (1907) records 3 cases of spontaneous lactation in unmarried nursemaids who repeatedly suckled their charges. Other causes mentioned are pineal tumours (Oestreich and Slawyk, 1899), disease of the ovary, testis and suprarenal cortex (Krestin, 1932), post-encephalitic parkinsonism (Riese, 1928) and acromegaly (Davidoff, 1926); galactorrhoea may also be a symptom of the menopause. Chiari *et al.* (1855) and Frommel (1882) described a condition of utero-ovarian atrophy associated with persistent lactation and amenorrhoea. This condition, however, appears after childbirth and is directly related to parturition, and further descriptions of the Chiari-Frommel syndrome have recently appeared in the literature (Gilbert, 1941, and Mendel, 1946). Persistent lactation not associated with pregnancy is a less common entity. Krestin (1932) describes 2 cases of spontaneous lactation associated with enlargement of the pituitary in non-pregnant, non-acromegalic women.

An interesting syndrome of spontaneous galactorrhoea, amenorrhoea and low urinary FSH excretion has been described by Forbes *et al.* (1954) and by Argonz and del Castillo (1953). Forbes *et al.* investigated 15 non-acromegalic women who presented with this syndrome. Of these 15 patients, 9 had never been pregnant, some had irregular periods, but the majority experienced amenorrhoea. Nearly all had some degree of obesity, hirsutism and seborrhoea and all but one exhibited a low urinary FSH excretion. Failure to menstruate following the administration and withdrawal of progesterone demonstrated a lack of endogenous oestrogen in the majority of cases. A striking feature of the series was the high incidence of pituitary tumour. In 8 of these 15 patients there was evidence of a tumour, and in 3 it was proved by biopsy to be a chromophobe adenoma of the anterior pituitary gland. These authors further studied lactation in nursing mothers and concluded that normal lactation was not due to the absence of FSH, which occurred in their group of nursing mothers in slightly higher amounts than in a group of non-lactating mothers. Nor did they feel that it was due to lack of oestrogen alone, because some nursing mothers have regular periods, and they maintain that neither the menopause nor castration cause lactation. These authors quote Cushing (1932-33), who postulated that the cells in the chromophobe adenomata are always pre-eosinophilic in type and, since lactation occurs in acromegaly, they feel that the syndrome is due to pituitary dysfunction whereby a specific hormone is produced in excessive amounts. Argonz and del Castillo (1953) express the view that spontaneous galactorrhoea is pituitary or hypothalamo-pituitary in origin, resulting in eosinophilic hyperfunction of the anterior pituitary. They feel that the condition is not primarily gonadal since, at the menopause and after castration, the urinary FSH is increased. The oestrogen insufficiency therefore resulted from lack of pituitary stimulation. They were, however, unable to detect any upset in the hypothalamus, nor were they able to demonstrate any evidence of a pituitary tumour in these cases, and substitution therapy did not influence the galactorrhoea.

SUMMARY

A case is recorded of a married woman aged 35 years who presented with the features of obesity, galactorrhoea, menstrual irregularity and sterility, not related to pregnancy or acromegaly. The literature bearing on the differential diagnosis is reviewed. The mechanism of this condition is not known but current views on this aspect of endocrine dysfunction are discussed.

We wish to thank Dr. M. E. L. Tonkin, Medical Superintendent Coronation Hospital, for permission to publish this report.

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Red Cross War Memorial Children's Hospital, Rondebosch, Cape. The next meeting in the Postgraduate Seminar Series of lectures under the auspices of the University of Cape Town Department of Child Health will be held in the lecture theatre of this Hospital on Wednesday 2 April at 5 p.m. Speaker, Dr. A. B. Murray; subject, 'Normal and abnormal formation of red blood cells in childhood'. All medical practitioners are welcome.

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EDITORIAL

TREATMENT OF ACCIDENT CASUALTIES

An article by Prof. J. H. Louw, of Cape Town, is published this week (page 337) dealing with the pathology and treatment of wound shock. Although the early care of the injured gets less consideration than its due, the subject is of first-rate and fundamental importance, and the principles of dealing with the injured should be widely disseminated. In South Africa more than half a million people are treated every year for injuries of various sorts and, of these, 7,500 lose their lives. For the most part accident victims are otherwise healthy, and the permanent disablement or death of a man with a dependent family is a serious loss to the community. Quite apart from the loss of life, the 'loss in working time, production, wages and compensation due to the subsequent illness of trauma' is enormous.

There is no fundamental mystery regarding the pathology of wound shock, and the aetiology is unhappily only too obvious. The shocked patient is suffering from circulatory collapse, which occurs quite simply when the volume of blood in effective circulation is sufficiently reduced either by haemorrhage, which may be on the surface or into the tissues, or by the loss of plasma, in burns and crushing injuries. A reasonably accurate evaluation of the shocked patient, which in effect is an estimate of the volume of whole blood or plasma irretrievably lost to the circulation (pooling or stasis in the capillaries is not an important factor in wound shock) can be made by a consideration of the blood pressure and the extent of the local injury, together with a study of the character of the pulse, the degree of vein filling, the colour and temperature of the skin, and the presence or absence of sweating. In this connection it is important to heed Professor Louw's warning that the clinician must not allow himself to be misled by the lack of external evidence of bleeding and absence of signs of shock. While it must be accepted that the blood pressure is not entirely reliable as an index of the blood volume it is none the less of considerable help in deciding whether or not transfusion is urgently necessary, and Professor Louw is of the opinion that a systolic figure of 100 mm. Hg is the threshold level below which transfusion is mandatory. The requirements of other cases must be judged on their merits, and if there is any doubt the patient s hould be trans-

VAN DIE REDAKSIE

NOODHULPBEHANDELING BY ONGEVALLE

'n Artikel deur prof. J. H. Louw van Kaapstad verskyn hierdie week op bls. 337 en gaan oor die patologie en behandeling van verwondingskok. Hoewel die vroeë behandeling van die beseerde persoon minder aandag geniet as wat dit toekom, is hierdie onderwerp van eersterangse en fundamentele belang, en die beginsels van die behandeling van beseerdes behoort wyd bekend gemaak te word. Meer as 'n halfmiljoen mense word elke jaar in Suid-Afrika behandel vir verskillende soorte beserings en 7,500 van hulle skiet daarby die lewe in. Gewoonlik is die slagoffers van ongelukke andersins gesond, en die permanente ongeskiktheid of dood van 'n man met 'n afhanklike gesin is 'n ernstige verlies vir die samelewing. En afgesien van die verlies van lewe is die ‚verlies aan werktyd, produksie, lone en kompensasie as gevolg van die daaropvolgende siekte van besering' geweldig.

Daar is geen fundamentele geheime oor die patologie van verwondingskok nie, en die etiologie is, betreurenswaardig, maar alte duidelik. Die skokpasiënt ly aan 'n ineenstorting van die bloedsomloop, wat heel eenvoudig voorkom wanneer die volume bloed in effektiewe omloop sterk genoeg verminder word deur bloeding, wat op die oppervlakte of binne die weefsels mag plaasvind, òf deur die verlies aan bloedwei, by brandwonde en verbryseling. 'n Redelik akkurate evaluasie van die geskokte pasiënt, wat eintlik 'n berekening van die volume van volbloed of plasma is wat die bloedsomloop onherroeplik verloor het (opdamming of stase in die haarvate is nie 'n belangrike faktor by verwondingskok nie), kan gemaak word deur die bestudeer van die bloeddruk en die omvang van die plaaslike besering, tesame met 'n studie van die aard van die pols, die mate van aarstuwing, die kleur en temperatuur van die vel, en die aanwesigheid of afwesigheid van sweetafskeiding. In hierdie verband is dit belangrik om professor Louw se waarskuwing ter harte te neem: die klinikus moet nie toelaat dat hy mislei word deur die gebrek aan uiterlike tekens van bloeding en die afwesigheid van tekens van skok nie. Dit moet wel aangeneem word dat die bloeddruk nie altyd die juiste weerspieëling van die bloedvolume is nie, maar dit is nietemin van veel waarde as daar moet besluit word of oortapping dringend nodig is al dan nie, en professor Louw meen dat 'n sistoliese druk van 100 mm. Hg die drumpelhoogte is waarbenede oortapping gebiedend noodsaaklik is. Die behoeftes van ander gevalle moet elk

fused, on the principle that the best treatment for impending shock is intelligent anticipation.

Basically, then, the treatment of severe shock is or should be a relatively straightforward affair; the patient has lost a large volume of blood or plasma, and may die if his blood volume is not speedily replaced. The overriding need in treatment is early, energetic and adequate transfusion, and anything short of this may well come into the category of ineffectual window-dressing. It is clear that if we are to improve the prognosis of wound shock, every effort must be directed towards solving the problem of ensuring the availability of adequate transfusion facilities at the scene of an accident. In this connection Professor Louw has made an eloquent appeal for the organization of an accident service on a country-wide basis, so that the experience gained in wartime, where immediate transfusion saved countless lives, can be translated to the requirements of civilian life. He makes the charge, and it is a serious one indeed, that despite our present-day knowledge of the basic therapeutic needs in traumatic shock, 'the profession has apparently not learnt this lesson and consequently failed to put its weight into the problem of improving the care of the wounded during the earliest phase'. It must be accepted that resuscitation can be given on the farm, at the roadside, in the backyard of a house, and in a properly equipped ambulance, just as effectively as in a hospital, and lives would be saved if on occasion removal to hospital was delayed for sufficient time to allow of the local setting up of an intravenous infusion. Since plasma, plasma substitutes and giving sets are easily procurable and have proved their life-saving qualities, it would appear a reasonable forecast that, in the not distant future, the doctor who arrives at the scene of an accident without the equipment necessary for immediate transfusion will, under certain circumstances, be regarded as failing to apply reasonable skill and care in the handling of the patient.

Last year[1] we drew attention to the importance of effective treatment of casualties at the scene of the accident. Professor Louw[2] has now spotlighted the glaring need in calling for a more dynamic approach to the problem of providing the country with an efficient accident service. Recommendations such as the establishment of surgical flying squads and blood banks or donor panels in country towns, and a helicopter ambulance service, deserve the attention of the State, and it is incumbent on the profession to support this appeal by bringing pressure to bear on the Provincial Administrations. A surgical flying squad, of which we have an example in the obstetrical flying squad of the University of Cape Town,[2] should be based on hospitals possessing properly equipped accident departments, and staffed by personnel trained in traumatic surgery. These squads should be considered as mobile appendages of such hospitals, and should not function as semi-autonomous units. The conception of a helicopter ambulance service is an intriguing one[3] and, in the light of its proven value in the Korean campaign and in view of the vast sparsely populated areas of

op eie meriete behandel word, en as daar enige twyfel bestaan of die pasiënt 'n bloedoortapping nodig het, moet ons die beginsel hou dat intelligente antisipasie die beste behandeling vir dreigende skok is.

Basies is, of behoort die behandeling van ernstige skok dus 'n betreklik eenvoudige saak te wees; die pasiënt het 'n groot volume bloed of plasma verloor, en hy kan sterf as sy bloedvolume nie spoedig weer tot op hoogte gebring word nie. Die eerste vereiste by die behandeling is vroeë, kragdadige en genoegsame oortapping, en al wat hierby te kort skiet kan bestempel word as ondoeltreffende ‚venstersversiering'. Dit is heeltemal duidelik dat as ons die prognose by verwondingskok wil verbeter, ons al ons kragte moet wy aan die oplos van die probleem om te verseker dat doeltreffende oortappingsfasiliteite beskikbaar is by die plek waar die ongeluk gebeur. In hierdie verband het professor Louw 'n kragtige beroep gedoen vir die organiseer van 'n ongevalle-diens op landswye skaal, sodat die ondervinding wat tydens die oorlog opgedoen is, toe tallose lewens deur onmiddellike oortapping gered is, ook toegepas kan word op die burgerlike behoeftes. Hy verwyt ons—en dit is 'n ernstige aanklag—dat, ten spyte van ons hedendaagse kennis van die basiese terapeutiese behoeftes by beseringskok, ‚die mediese beroep klaarblyklik nog nie hierdie les geleer het nie en gevolglik versuim het om sy deel by te dra tot die kwessie van hoe om die vroeë behandeling van ongelukslagoffers te verbeter'. Daar kan tog nie aan getwyfel word dat 'n pasiënt weer net so doeltreffend bygebring kan word op die plaas, langs die pad, in die agterplaas van 'n huis, as in 'n hospitaal nie, en baie lewens kan gered word indien die vervoer na die hospitaal in sommige gevalle vertraag word sodat daar tyd genoeg is om op die ongeluksplek binneaarse oortapping in te stel. Aangesien plasma, plasmavervangers en toedieningsappartae maklik bekombaar is en hul lewensreddende hoedanighede reeds op die proef gestel is, blyk dit 'n redelike vooruitsig te wees in die nabye toekoms dat dié dokter wat sonder die toestelle nodig vir onmiddellike oortapping op die ongeluksplek aankom onder sekere omstandighede daarvan beskuldig kan word dat hy nie redelike en verwagbare vaardigheid en versigtigheid toegepas het die behandeling van sy pasiënt nie.

Verlede jaar[1] het ons die aandag gevestig op die belangrikheid van doeltreffende behandeling van ongevalle op die plek waar die ongeluk plaasvind. Professor Louw het nou die kalklig gespits op die skreiende nood in sy beroep vir 'n meer dinamiese houding teenoor die probleem van die voorsiening van 'n doeltreffende ongevalle-diens vir die land. Aanbevelings soos die daarstelling van chirurgiese, blitspatrollies en bloedbanke of skenkersnaamlyse in die platteland se dorpe, en 'n helikopter-ambulansdiens, verdien die aandag van die Regering, en dit is die plig van die mediese beroep om hierdie beroep te ondersteun deur hulle te laat geld by die Provinsiale Administrasies. 'n Chirurgiese snelspan—as voorbeeld dien die verloskundige span van die Universiteit Kaapstad[2]—moet daarop berus dat die betrokke hospitale oor behoorlik toegeruste ongelukkeafdelings beskik en dat hul personeel opgelei is in beseringschirurgie. Hierdie spanne moet beskou word as mobiele vertakkings van sulke hospitale, en behoort nie as halfselfstandige eenhede te werk nie. Die idee van 'n helikopterambulansdiens is baie interessant, en met die oog op die waardevolle diens wat so 'n instelling in die Koreaanse oorlog gelewer het, en ook met die oog op die enorme dun-

our country, it would appear to be the ideal means of transport for the accident victim in remote places.

It is to be hoped that there will be no undue delay in implementing Professor Louw's recommendations for a new and greatly improved Union-wide accident service, which is now an urgent necessity.

1. Editorial (1957): S. Afr. Med. J., 31, 468.
2. Hagberg, C. J. (1956): *Ibid.*, 30, 1140.
3. Edwards, A. G. (1958): Lancet 1, 470.

bevolkte streke van ons land, blyk dit die ideale vervoer te wees vir ongeluksgevalle in afgeleë dele.

Ons hoop dat daar nie 'n onnodige vertraging sal wees nie met die verwesenliking van professor Louw se aanbevelings vir 'n nuwe en grootliks verbeterde landswye ongevallediens. Dit is tans 'n dringende behoefte.

1. Van die Redaksie (1957): S. Afr. T. Geneesk., 31, 468.
2. Hagberg, C. J. (1956): *Ibid.* 30, 1140.
3. Edwards, A. G. (1958): Lancet 1, 470.

THE PROBLEM OF WOUND SHOCK AND RESUSCITATION IN RELATION TO THE SURGERY OF REPAIR*

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Repair is defined as restoration to a sound state after injury. Before discussing the subject shock, I feel it is pertinent to consider briefly the problem of injuries in general and their impact on the welfare of the State.

The term injury is usually associated with physical violence, and includes both the local and general effects of trauma. Grant and Reeve[30] have coined a more appropriate term 'the illness of trauma' to cover the whole picture of the illness, local and general, from the moment of injury to complete recovery. This illness includes the following:

1. Local tissue damage, usually a wound or a burn. This alone may be responsible for considerable disablement and, if a vital organ such as the brain is involved, for the death of the patient.

2. The state of shock. This follows every injury of any magnitude and accounts for more than half of the fatalities in accidental injuries[4,29,30,39,44] and for fully three-quarters of the deaths in burns.[11,23]

3. Widespread reactive changes with profound endocrinal, metabolic and hematological disturbances. These processes, which have been called *the metabolic response to trauma*[33,45] are closely related to the degree and duration of shock and have an effect on recovery from shock, wound healing, susceptibility to infection, incidence of complications and the whole question of morbidity after injury.[7-10,19,26,45]

In civilian practice *accidents* are the principal cause of fatal and disabling injuries. In South Africa more than half a million people are treated every year for injuries of various sorts. About 40% of these occur in our homes, and the rest, including most of the serious injuries, are due to industrial and road accidents, both of which have shown an alarming increase in recent years.[34,41] Since 1950 our road injuries have increased by over 50% and industrial injuries by 30%.

The result is that in our European population accidents rank only after circulatory diseases and cancer as a major cause of death.[2-4] About 7,500 people of all races lose their lives every year as a result of accidents. Many of them are men in the prime of life; indeed accidents rank as killer number one in males up to the age of 40 years.

The loss of life, however, represents only a small proportion of the enormous loss in working time, production, wages and compensation due to the subsequent illness of trauma.[41] Approximately 10-15% of the injured suffer some degree of permanent physical disability and many more spend long periods in hospital and in receiving treatment. Industrial accidents alone are responsible for the loss of 20 million man-days, costing the country approximately 20 million pounds every year. This is equivalent to the permanent loss of 3,200 persons or 50,000 workers stopping work for a year, or a paid holiday for every worker for 11 days every year.

The magnitude of the problem is obvious and its solution a question of national importance. We as a profession can contribute a great deal towards prevention but our main function is to lessen mortality and morbidity by assisting Nature in restoring the body to a sound state after the ravages of trauma, i.e. by the surgery of repair.

WOUND SHOCK

Many words whose significance is well understood are difficult to define,[12] and Wound Shock is one of them. In trying to understand the true meaning of the term it is important to appreciate that *every injury of any magnitude is followed by shock*. It is, therefore, correct and proper to assume that shock is present in any person who has sustained a major injury. In practice, wound shock is usually interpreted as a state of circulatory collapse after injury.[38] 'All we know when we are called to the patient's side is that he has been injured, that he has collapsed and that he lies apathetic with an ashen grey face and cold clammy hands'.[38] The initial diagnosis is essentially clinical and, having made it, it is then our duty to ascertain the exact cause of the collapsed state and institute appropriate treatment.

The Mechanism of Shock. What are the causes of shock?

This is best understood by considering what factors are responsible for an adequate circulation. Briefly, these are the action and force of the heart, the volume and viscosity of the blood and the peripheral resistance of the arterioles. Interference with any one or more of these may produce a shock-like state.

For many years it has been known that the essential feature in traumatic shock is a reduction in the volume of blood in effective circulation. This may be due to actual loss of blood

* Opening paper in Plenary Session on the Surgery of Repair, South African Medical Congress, Durban, September 1957

or plasma or to a sudden increase in the capacity of the vascular bed while the actual blood volume remains unchanged or only slightly diminished. Thanks to the research work of Grant and Reeve[30] during World War II, we now know that actual reduction of blood volume is of the greatest importance. In the case of wounds, vast quantities of *whole blood* are lost, not only to the exterior but also into the tissues.[7,17,29,30] The latter is of particular importance because it is not obvious; e.g., a man can bleed to death into the thigh muscle[5] around a fractured femur without any external wound.[38] In the case of burns there is loss of *plasma* to the exterior and also into the tissues and in extensive burns this may amount to many litres.[7,11] *Today, we realize that the most important single factor responsible for traumatic shock and early death following injury is loss of whole blood from wounds and plasma from burns.*[7] In general, a sudden loss of 25% of the blood volume endangers life, and recovery without transfusion is unlikely with losses of 40% and more.

A sudden increase in the capacity of the vascular bed with 'pooling' of appreciable quantities of blood is of secondary importance. It may be due to:

(*a*) Neurogenic Factors. These produce a transient increase in vascular capacity and a shock-like condition which responds rapidly to rest, relief of pain, warmth and other simple resuscitative measures.

(*b*) Vasogenic Factors. These may play a role in crush injuries and abdominal trauma with peritonitis and are responsible for an increase in the capacity of the vascular bed at a somewhat later stage[7,17] They cause serious depression of arteriolar tone which does not respond to transfusion alone, but may be helped by potent vasopressors such as nor-adrenaline.

The Assessment of the Shocked Patient

Oligaemic (hypovolemic, haematogenic) shock, *must be assumed to be present in all cases that have sustained a major injury*, particularly where there has been much blood loss or tissue damage. Subsequent assessment depends on the following:

1. *Clinical evaluation.* This is obviously of importance, but clinical features may be conspicuous by their absence in the early stages after the injury or in cases of so-called subclinical or latent shock.[7] Among the various clinical features, the blood pressure is probably the best 'index of the blood volume' but it is not entirely reliable.[29,30] In general, if the systolic pressure of a normal adult has dropped below 100 there has been a loss of more than 2½ pints of blood.[30] It must be remembered, however, that the blood pressure is often unaffected by smaller losses and in children and young adults there may even be a rise of blood pressure with losses as great as 20% to 30% of the blood volume.[38] Occasionally patients manifest periodic rises and falls in the blood pressure and the fact that the patient might have been hypertensive also clouds the issue.[12] However, as indicated in Table I, the height of the systolic pressure affords some indication of the blood volume and the figure of 100 systolic can be taken as the critical level below which urgent treatment becomes obligatory. It is obviously better to correlate the blood pressure with other circulatory signs and clinical features which result from the injury. In this way various combinations of signs may be grouped together to form distinct patterns.[48] These, with their interpretations are summarized in Table II.

SHOCK – CLINICAL PATTERNS

CAUSE	SYSTOLIC B.P. (mm. Hg.)	PULSE RATE	SKIN	BLOOD VOLUME (per cent of normal)	PATTERN
A. External fluid loss (blood or plasma)	> 100	< 100	Warm extremities, good colour	> 80	Nearly normal
	< 100	> 100	Cold extremities, pale face	< 70	Cold hypotension
	< 70	Impalpable	Very cold and pale extremities and face	< 60	Extreme cold hypotension
	> 100	> 100	Cold extremities, pale face	70–80	Cold tachycardia
B. Neurogenic	> 140	< 100	Warm or cold extremities, good or pale colour	Adults > 80 Children < 70	Post-traumatic hypertension
	< 100	< 70	Cold extremities, pale face	Normal or almost normal	Vasovagal reaction

1

THE BLOOD PRESSURE AND BLOOD VOLUME

BLOOD PRESSURE	BLOOD VOLUME
> 140	> 80%
100 to 140	70 to 80%
70 to 100	60 to 70%
< 70	< 60%

2

2. *Evaluation from the extent of the local injury.* More accurate assessment can be made from the extent of the local injury, which affords 'an index of the blood loss'[7-9,13,19,29,30] This applies to both wounds and burns. In general, there is a close correlation between the local injury and the degree of shock—the larger the injury, the greater the blood loss and hence the more severe the reduction in blood volume.[7,30] In this connection it is important to repeat that fluid, blood or plasma, may be lost into the tissues without external loss and this loss must obviously be taken into account in assessing the degree of shock.

When the blood loss is entirely external, the amount of blood that has been absorbed by the clothes, dressings etc. affords a rough idea of the losses. In surgical operations, for instance, blood loss is estimated by the amount of blood that has been absorbed by the swabs and towels that have been used. We make use of this in certain circumstances by weighing the swabs before and after use and so obtaining a fairly accurate assessment of the loss. With experience one can learn to assess how much blood a swab contains by noting the amount of staining that is present.

In the case of open accidental wounds the size of the wound gives a fairly accurate index of blood loss. Using the hand as the unit of volume, it is in this way possible to classify wounds in terms of severity of blood loss.[30] The volume of an open hand or closed fist amounts to half a litre. Therefore a wound the size of an open hand in the case of superficial wounds or of a closed fist in the case of deep and lacerated wounds will be associated with the loss of about half a litre of blood. In general, wounds the size of one hand are not accompanied by any recognisable signs of shock, those of 2 hands are associated with moderate shock, those of 3 with severe shock, and those of 4 with a profound degree of shock.[38]

With closed injuries of the extremities an estimate of the amount of tissue damage can be made from the degree of

soft-tissue swelling and also from the situation and number of fractures that are present.[7,9,38,48] With experience it is possible to estimate the degree of swelling in terms of hand volumes and to classify the injury accordingly. Single fractures are associated with a blood loss of up to 1 litre, although a fractured femur may be accompanied by a loss of as much as 2 litres. With two fractures the blood loss amounts to 1-2 litres and with three or more fractures, more than 2 litres of blood are lost.[48]

With burns, the extent of body surface involved is equally important in assessing the degree of shock. This extent may be judged from Berkow's rule of nines. In adults, burns of less than 10% are associated with only slight shock, those of 10-15% with moderate shock, and those of 15% and over with severe shock.[11]

With penetrating wounds of the abdomen or chest the extent of the injury cannot be gauged by external examination and we have to rely on other signs.[7] In head injuries blood loss is usually small. This is important, because if a patient who has sustained a head injury is severely shocked, it is essential that other lesions should be sought for.

3. *Evaluation from changes in the composition of blood.* This is done by estimating the haemoglobin or preferably the haematocrit reading of a sample of blood. It must be remembered, however, that changes in haemoglobin or haematocrit concentration are often delayed for several hours and therefore may not be helpful in the early diagnosis of shock. Indeed, with wounds the readings are so fallacious that no reliance can be placed upon them.[30] With burns, on the other hand, the degree of haemo-concentration is of some value after a few hours and is most helpful in assessing the subsequent progress of the patient and the effect of treatment.

TREATMENT

In the treatment of wound shock *time is the most important factor*.[7,48] With wounds, approximately half the total blood loss that will occur takes place in the first 3 hours after wounding and massive quantities may be lost in a matter of minutes with sudden death if a major vessel is cleanly severed.[9] With burns, one-third of the total plasma that will be lost, is lost in the first 6-8 hours after burning, and in extensive burns as much as 20% of the blood volume may be lost in 15 minutes.[47] Delay is chiefly responsible for so-called irreversible shock, and injuries therefore present an urgency which seldom exists in other surgical conditions.

Blood Transfusion and Shock

The prime need in the treatment of wound shock is transfusion, which must be early, energetic and plentiful. Transfusion takes precedence over any local treatment except temporary splinting of fractures, covering of exposed wounds and arresting of violent haemorrhage. All other measures must be completely subordinated to transfusion.

As far as possible, blood loss from wounds, open or closed, must be replaced with cross-matched whole blood of the same group. In emergencies, low-titre group-O blood may be used, and if blood is not available plasma or serum should be transfused.[1,5,7,10,46,48] Where the blood loss does not exceed 1 litre, plasma alone may suffice.[38] When larger quantities of blood are lost then not more plasma than 1 part for every 2-3 parts of whole blood should be used. Plasma substitutes are, on the whole, unsatisfactory for the treatment of shock which is due to loss of whole blood.[38] They can restore blood volume temporarily but have many disadvantages; for instance, they interfere with compatibility, they do not carry oxygen, they do not provide any energy, they interfere with the clotting mechanism and their administration may be associated with severe reactions. However, if there is a desperate emergency and no blood or plasma can be obtained, plasma savers may have to be given, because desperate situations demand desperate remedies. In such cases it would be unwise to give more than 1 litre of the substitute.

Plasma loss from burns or crushing injuries must be corrected by transfusion of plasma or plasma substitutes. Plasma is the ideal fluid to be given. Serum is almost as good. When neither plasma nor serum are available, plasma substitutes may be given with a fair degree of safety and, in extensive burns, as much as 2-3 litres may be administered.[48]

As already mentioned, transfusion if it is to be effective must be early, energetic and plentiful.

Immediate transfusion is necessary in cases with a marked degree of circulatory failure if life is to be saved, and it is unwise to delay transfusion in any case with a blood pressure below 100 mm. systolic that does not recover promptly in the head-down position.[29,30] Any patient who has lost more than 1-1½ litres of blood must be transfused, especially if he has to be submitted to surgery or if further bleeding is likely to occur.[7]

Energetic transfusion is necessary in all cases with extensive injuries and a blood pressure below 100 mm. systolic. During World War II we often used to run in the first litre in 5-15 minutes and subsequent litres in 20-30 minutes. If necessary, 2 or more intravenous drips may be put up and pressure apparatus used. In this connection I would like to mention that intra-arterial transfusion has not much to commend it and is fraught with so many dangers that it is best avoided.

Plentiful amounts are required, particularly where there is continued loss of blood or plasma. The ultimate object is near-complete restoration of blood volume to render the patient fit for operation and to lessen the 'illness of trauma' and the incidence of complications. The immediate object is to restore the blood pressure to at least 100 mm. systolic.[30] Once this has been achieved, approximately 2 more pints of transfusion fluid will be required, even if there is no further loss. We frequently transfused amounts up to 10 pints during World War II and during the Korean War, 15 - 30 pints were often given to the critically wounded within the first 24 hours of wounding.[40] Indeed, in Korea massive transfusions up to 30 or 50 pints were sometimes given.[3] Incidentally, when such massive quantities of citrated blood are used there is a grave danger of citrate poisoning, which may be prevented by injecting calcium gluconate.

Ancillary Measures

These include relief of pain, proper posturing, warmth, the administration of fluids by mouth, the use of pressor drugs and, on rare occasions, the use of cortisone. *Relief of pain* is the most important. Morphine is the most useful analgesic but must be used with caution. It should be given intravenously since in the state of shock it is absorbed only very slowly from the subcutaneous tissues. *Elevation of the feet* is useful for assisting the cerebral circulation and may be all that is required to counteract the collapse of neurogenic shock. *Warmth* may be beneficial but excessive

heat which causes vasodilatation must be avoided at all costs. *Oral fluids* in the form of warm tea are of doubtful value and must definitely not be given to patients with abdominal injuries nor to those with a tendency to vomit nor where early operative intervention is contemplated. *Vaso-pressor drugs* are best avoided. In oligemic shock they may do harm and in neurogenic shock they are unnecessary. *Nor-adrenaline* is of value in cases with severe infection or extensive crushing of tissue where vasogenic factors play a role in the picture of shock[17] but this is seldom seen soon after an injury. *Cortisone* has no place in the treatment of traumatic shock *per se* and is indicated only in patients who have been receiving steroid therapy for some pre-existing condition. When, however, the patient manifests the so-called hypo-adrenal response to trauma, the administration of cortisone may be life-saving.

Operation

Operative intervention must be regarded in some cases as an essential part of the treatment of shock.[17,30] When there is severe and rapid blood loss from a divided major vessel, immediate intervention to arrest the haemorrhage is obviously a necessity because shock will not be controlled until the haemorrhage has been checked. Abdominal injuries, for instance, respond poorly to resuscitation until the source of bleeding has been found and controlled. Similarly, some patients with sucking chest wounds or very large limb injuries may have to be operated on at once if their lives are to be saved, and operation and transfusion for resuscitation may have to be carried out simultaneously.[7]

In general, however, the patient should be operated upon as soon as, but not before, he has been adequately resuscitated. Continual transfusion during the operation can enable even those with great blood loss to survive the operation with safety.

Controversial measures

In concluding my remarks on the treatment of wound shock, there are some controversial measures which may be mentioned:

Firstly, the contribution of bacterial toxins to the circulatory failure of oligemic shock. Fine and his associates in 1954 demonstrated bacteria in the blood stream and the tissues of animals subjected to haemorrhagic shock and showed that both bacteraemia and some of the clinical and haemodynamic features of the condition could be prevented by preliminary treatment with broad-spectrum antibiotics.[17] In clinical practice this bacterial factor is probably of very small significance, particularly where the patient has been resuscitated early in the course of shock. However, when hypotension is prolonged or when severe muscle damage with infection is present, a shock-like state may persist until the infection is brought under control or the damaged muscle excised.

Secondly, the use of drugs to protect the patient from shock. In recent years chlorpromazine has been shown to protect animals from the effects of graded haemorrhage and this drug has therefore been suggested for the treatment of patients suffering from wound shock. At present, however, the clinical use of such techniques as the 'lytic cocktail' does not appear to have any advantage over more conventional methods of prevention and treatment of shock.[7,17]

And lastly, there is the question of cooling. The rationale of this method of treatment is to reduce harmful excessive reactions on the part of the injured patient. At this stage it is fair to say that there is not enough evidence to warrant the use of hibernation in the treatment of severe injuries, with the possible exception of certain head injuries.[17]

THE MANAGEMENT OF SHOCK IN RELATION TO ACCIDENTAL INJURIES IN SOUTH AFRICA

At the beginning of this address, I indicated to you the great wastage of man power in South Africa caused by accidental injuries, particularly serious injuries occurring in our industries and on our roads. It was also pointed out that shock and the so-called illness of trauma which follows on shock were responsible for the majority of fatal injuries and for a great deal of subsequent disablement and loss of working time.

Then I have made an attempt to indicate what our modern concept of shock is; viz. that it is primarily due to blood loss and that the proper treatment of shock is, in the first place, replacement of the blood that has been lost. Our experiences have shown that blood replacement plays a vital part, not only in the treatment and prevention of shock, but also in allowing more adequate primary surgery and in influencing the whole pattern of the illness which follows on the injury.[7] In this way proper treatment not only saves lives but also shortens the convalescence which follows an injury and so saves man-power.

The recent wars have shown that if modern facilities for the treatment of the wounded can be made immediately available to the patient, the death rate can be considerably lessened.[20,15] Thus there has been a steady and constant improvement in the salvage figures from less than 40% saved in World War I to 97% saved in the recent Korean War.[18,19,14]

The lessons learnt during these wars have proved a tremendous boon to mankind. Their application to civilian injuries since the war has enabled us to save many more lives than before and to reduce the morbidity of accidental injuries considerably. Thus, in Birmingham, where a first-rate accident service has been established, the results of the treatment of civilian casualties are even better than those obtained during the Korean campaign.[22,27] Today it is generally accepted that if the patient can only be brought alive to a properly equipped hospital, his life will probably be saved.[25-27] This was well illustrated by a recent train disaster in the Western Cape.[18] Two trains carrying office workers home after the day's work came into a collison, in which 93 were injured. Of these, 18 died on the spot while 75 were taken to 2 neighbouring hospitals, one our University's teaching hospital and the other a suburban hospital. Although 20 of the patients admitted were seriously injured, with multiple fractures, etc. there were no further fatalities. One cannot do better than save 100% of the injured and so far as salvage of life is concerned, it would appear that our hospitals are indeed coming close to the ideal.[18]

Let us now consider briefly what were the main lessons learnt from recent wars and how we can apply these lessons to civilian injuries.

1. The *first* lesson to be learned from wartime experience comes from the forward field surgical units or field hospitals, which were placed close to the front line and within easy reach of war casualties. In these forward surgical units unhurried and perfectly organized resuscitation was regarded

as the essential step in the treatment of the injured and all facilities for the treatment of shock were immediately available.[15,16,27]

The counterpart of this in civilian practice is to be found in the establishment of accident hospitals and departments. As already mentioned, such an accident hospital organization has been established in Birmingham and has proved to be a great success. I should like to pay tribute to Mr. Gissane, who is with us today, for the most valuable work that he has done as Director of this hospital. As he points out, such a hospital must adopt a completely new tempo and quality of hospital organisation.[26] It should provide a round-the-clock service with skilled surgical teams, resuscitation units, and all necessary ancillaries. It consists essentially of a central accident hospital with full-time consultant staff linked peripherally with the casualty departments of neighbouring hospitals and centrally with a general hospital, preferably a teaching hospital, which has modern orthopaedic, plastic and other facilities.[20,26] To be really efficient the accident hospital should be not more than 70 miles, preferably only 40 miles, from any serious accident, and such a service is justified only in densely populated areas, i.e. in cities with a population of approximately 200,000 and an immediate neighbourhood population of about 2,000,000.[20,27] From the economic point of view, the hospital should treat *not* less than 25,000 patients per year.

Now let us consider the situation in South Africa and see what can be done about the establishment of such an accident service. On looking at a map of our country, it immediately becomes obvious that it is difficult to fulfil the requirements for an efficient service because of our vast distances and sparse population. We have only 6 cities with populations of 200,000 or more and only 5 with populations between 100,000 and 200,000.[6,34]

The Witwatersrand area is the only area in which the population is sufficiently dense to warrant a proper accident hospital. In this area we have Johannesburg with its population of 1 million and a population of about 2½ million in the surrounding area within a radius of 40 miles. In this area we also have Germiston with a population of over 200,000 and Pretoria with a population of a third of a million. Furthermore it is a great industrial centre and more than a quarter of all our industrial accidents occur here. The stage is therefore set for the establishment of at least one and probably two proper accident hospitals based on the pattern of the Birmingham Accident Hospital.

The next big centre to be considered is Cape Town. Here the city itself has a population of almost three quarters of a million and the hospitals in the area drain a population of over a million. Although an accident hospital as such will not prove an economic proposition, the establishment of a big accident department attached to the teaching hospital of the University of Cape Town at Groote Schuur now becomes an urgent necessity.

Durban has a population of over half a million and drains an area with a very large Native population. Here a large accident department attached to the teaching hospital is also an obvious necessity. Port Elizabeth—though its population is still small—about a quarter of a million—is a rapidly developing industrial centre and the establishment of an accident department connected with one of the larger hospitals would even at this stage seem highly desirable. Finally, there is Bloemfontein with a population of only just over 100,000 but centrally situated. An accident department to drain the central part of this large country of ours could profitably be established there.

The rest of our country is too sparsely populated to warrant an accident service on the above scale and we must therefore rather aim at improvement and elaboration of the existing facilities. The improvements should include the following:

(*a*) Improved design and equipment of all casualty departments, with provision of facilities for the treatment of shock on the spot.

(*b*) More attractive salaries for our casualty officers which would draw men of greater experience and with a surgical leaning.

(*c*) The establishment of proper orthopaedic and plastic departments, a burns unit, and facilities for rehabilitation, in all our larger hospitals.

2. The *second* lesson to be learnt from the wars concerns the immediate availability of blood and plasma for resuscitation of casualties on the spot where they have sustained their injuries. It has already been pointed out that delay in commencing resuscitation is the greatest enemy to life. The best surgical skill and hospital equipment in the world are of no use if the race with time is lost.[7] Our people in South Africa are scattered over an enormous territory with no abundance of fully equipped hospitals. There is obviously a great need for resuscitative centres and facilities at various vantage points in the country.

During World War II in the Western Desert, where the huge distances and rapidity of movement created great difficulties in supplies and evacuation, the situation was somewhat similar to that which obtains in South Africa. In the war the responsibility for early resuscitation often rested heavily on the battalion medical officer. The immediate availability of plasma or blood at regimental aid-posts and the efficiency of the battalion M.O., proved to be vital factors in the salvage of lives.[32] Thanks to the excellent organization of the 8th Army Transfusion Service a standard was achieved whereby not only plasma but also whole blood was provided to advanced dressing stations and even to regimental aid posts.[35,36] In desperate cases, transfusion was commenced on the battlefield and continued in the ambulances during evacuation.

In considering fatal accidents in South Africa, it immediately becomes obvious that most of the deaths on our roads and in our railway, flying, mountaineering and recreational accidents occur during the period immediately after the injury and before admission to hospital. In the railway disaster referred to before, 20% of the injured succumbed at the site of the accident and of these more than half died of shock. We might well ask ourselves whether those persons who died of shock might not have been saved had proper facilities been immediately available. The recent wars have shown that future developments in salvage of life will depend not so much on perfection of the already near-perfect hospital treatment but on attention to the patient on the spot.[32] In spite of the tremendous advances in the hospital management of civilian casualties, our profession has apparently not learnt this lesson and has consequently failed to put its energies into the problem of improving the care of the wounded during the earliest phase.[42]

The responsibility rests squarely on the shoulders of the general practitioner, who is usually the first doctor to reach the site of an accident. He should be thoroughly proficient

in the recognition of shock, the assessment of blood loss, and the early care of the wounded. Many a battalion medical officer transfused his patients in the heat of battle. There is no reason why the general practitioner cannot do the same at the scene of an accident. Dried plasma and plasma substitutes which can be kept indefinitely and transported with ease are readily procurable and have proved their worth as life-saving measures. *Every doctor should have a supply of these, together with suitable giving sets, in his car as well as in his surgery.*

While very early transfusion of plasma will no doubt save many lives, it has already been pointed out that adequate treatment of wound shock demands whole blood in plentiful amounts. Thanks to the magnificient services of our blood transfusion organizations with their headquarters at Cape Town, Port Elizabeth, East London, Johannesburg and Durban, blood can now be provided in most parts of the Union.[47] However, the areas served are necessarily very large. The Western Province service, for instance, covers half the Cape Province. Here the difficulty of providing blood at short notice has been partly overcome by the establishment of branches with banks or donor panels in various Western Province towns.[47] Nevertheless, there are many areas where blood cannot be kept or obtained on the spot. In such cases, blood has to be transported from headquarters or from the nearest branch. This takes time and is burdensome and costly on account of the need to maintain the blood at a low temperature. The establishment of more donor panels in our country towns is an obvious step in rendering blood readily available and the ideal would be to have every town and village in South Africa provided with either a branch bank or a donor panel.

Other problems which arise in connection with the provision of blood on the spot concern the inherent risks attendant upon blood transfusion, and the need for grouping and compatibility tests. In emergencies low-titre group-O blood, which was used extensively during the wars, has to suffice, but in many instances it will probably still be most practical to treat the patient on the spot by the infusion of plasma or plasma substitute. There is no reason why dried plasma and plasma substitute should not be made immediately available even in the remotest parts of our country. Not only should every doctor have a supply at hand but plasma should be kept in every ambulance and train, in all large factories, at key points along the national roads, aerodromes, racetracks, mountain-club huts, fire stations and so on. This would obviously involve a tremendous organization, but it is a matter of national importance and deserves the attention of the State. Blood and plasma were made readily available during the wars and there is no reason why in a peace time organization a similar ideal cannot be achieved.

3. The *third* lesson to be learnt from the wars concerns the speedy evacuation of casualties to a hospital with continuation of transfusion, during transport if necessary. In World War II, ambulances with facilities for in-ambulance transfusion were always readily available and standing by to convey casualties to the nearest advanced surgical unit. The Korean war proved the value of helicopter evacuation, which made it possible to get all casualties back to a surgical unit within an hour of having been wounded.[3,26]

The safe transport of seriously injured patients to a hospital can only be effected by the provision of a mobile surgical unit, with staff who can continue the resuscitation during the journey. In Birmingham such a unit, with special stretchers and splints for comfortable travelling and all facilities for blood transfusion etc., has proved its value as a life-saving measure.[20,27] In Cape Town, we have now had 4 years' experience of a somewhat similar unit for obstetrical emergencies.[31] This *flying squad*, which was established from the University of Cape Town Students Hospital Rag Fund, operates from the hospitals which are attached to our medical faculty. It covers the whole of the Cape Peninsula and surrounding areas and can be called upon by any doctor or midwife faced with an obstetrical emergency. Usually the squad is on its way within 15 minutes of the call having been received. Many calls have come from our worst slums and in several cases, especially on the Cape Flats, it was necessary to carry the equipment, which always included a supply of blood, over a mile or more of sand and bush because the ambulance could progress no further. In some cases, it was necessary to transport the patient back over the same route, this time with intravenous drip in position. In the majority of cases the squad was faced on arrival with severely shocked and exsanguinated patients requiring immediate and energetic transfusion. The results of this work have been illuminating. In the first 200 calls there were only 6 deaths; 3 patients were dead when the squad arrived and the remaining 3 succumbed within 15 minutes of the arrival of the squad. The squad has more than justified its existence and it is high time for the service to be taken over by our Provincial Administration and extended to other branches of medicine. If this were to be done, properly equipped mobile surgical units built on the Birmingham pattern should be provided and attached to our larger hospitals, while all other ambulance units should carry blood, which can now be provided in convenient plastic containers suited for in-ambulance transfusion.

Furthermore, in our country with its vast distances, the possibility of air transport should be given serious consideration and the establishment of a helicopter ambulance service should receive our earnest attention. Helicopters have been tested not only in the wars but in other countries also in peace time and have already saved thousands of lives. Our country would be well-advised to try to improve upon the Australian 'flying doctor' service which has been using conventional aircraft, by setting up a similar organization equipped with helicopters.

Before concluding my remarks on the problem of resuscitation on the spot may I direct your attention to a few interesting speculations?

(*a*) Should specially selected laymen be trained in the discipline of blood transfusion so that the administration of plasma could become as routine as, for example, the injection of morphine in the treatment of the seriously injured?

(*b*) To what extent could bodies such as the National Occupational Safety Association and National Road Safety Organisation contribute to the immediate treatment of the injured?

(*c*) Could the services of flying, racing, mountaineering and other clubs be utilized to facilitate early transfusion and speedy evacuation?

(*d*) Should the Blood Transfusion Services enter the field of resuscitation more actively?

(*e*) Should the State supply standardized transfusion equipment, render blood and dried plasma readily available, and provide road and air transport for the injured?

As I see it, the problem hinges largely on the following two questions:

Firstly, what deaths are avoidable if, in the ideal state of organization, resuscitation can always be offered on the spot? Secondly, are the costs involved in providing the ideal state, justified?

The answers depend largely on the value we place on premature disablement and loss of life in terms of national economy. From the humanitarian point of view no effort is wasted and no expense too great; for, apart from the economic consequences, a serious accident is a major tragedy in the life and household of a person with a fit and healthy body.

SUMMARY

The problem of shock and resuscitation is considered in relation to accidental injuries in South Africa. Road and industrial accidents, which are increasing at an alarming rate, are generally responsible for the serious and fatal injuries which account for 7,500 deaths per annum and a great deal of ill-health and loss of man-power.

It is pointed out that every injury of any magnitude is followed by wound shock and that the essential feature is a reduction in the circulating blood volume due to loss of blood or plasma.

The diagnosis of shock is briefly considered in terms of clinical features, extent of the local injury, and changes in the composition of the blood. The close correlation between the size of the local injury and the degree of shock is emphasized.

It is pointed out that the prime need in treatment is transfusion, which must be early, energetic and plentiful, and time is the all-important factor in serious injuries. In practice this need may be met by:

1. Establishment of a comprehensive accident service with central accident hospitals on the Birmingham plan. This requires density of population and in South Africa is practicable probably on the Witwatersrand only. In Cape Town, Durban, Port Elizabeth and Bloemfontein, accident departments attached to general hospitals should be provided.

2. Improvement of facilities in the casualty departments of all our general hospitals.

3. Provision of blood, plasma and plasma substitutes 'on the spot'. In this connection the following suggestions merit consideration:

(*a*) Availability of dried plasma and plasma substitutes in all doctors' cars and surgeries, in every ambulance, train and factory, and at various points where accidents might occur.

(*b*) Establishment of branch blood banks or donor panels in all our country towns and villages.

(*c*) Provision of mobile surgical units with 'flying squads' and introduction of air ambulance services.

The possibilities of utilizing the services of specially trained laymen, the Blood Transfusion Services, and various organizations and clubs, are mentioned and it is urged that no effort should be spared in providing prompt treatment to all casualties.

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INDICATIONS AND LIMITATIONS OF HYPOTENSIVE DRUGS IN THE TREATMENT OF HYPERTENSION OF PREGNANCY *

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This survey was undertaken to determine the value of the hypotensive drugs in toxaemia of pregnancy. Because of lack of regular ante- and post-natal attendances we were unable to establish definitely whether our patients were suffering from toxaemia or whether the hypertension was due to other causes. The problem is further complicated because toxaemia may be superimposed on pre-existing hypertension or on chronic nephritis. From the therapeutic point of view it is important to differentiate because hypotensives appear to react differently in the different types of case.

Classification. Hypertension in association with pregnancy and labour occurs in about 5% of all pregnancies which progress beyond the 28th week. Hypertension in pregnancy may be classified as follows: (1) Pure pregnancy toxaemia (pre-eclamptic toxaemia), (2) essential hypertension, (3) essential hypertension with supervening pregnancy toxaemia, and (4) chronic nephritis. The significance of the classification is twofold; it enables one to decide whether hypotensive drugs are required and it assists one in prognosticating the foetal chances of survival.

For the purpose of this study any blood pressure above 140/100 mm. Hg was treated with hypotensive drugs; complications usually occurred above this critical level, though it is accepted from clinical experience that in uncomplicated essential hypertension hypotensive therapy is usually unnecessary if the blood pressure does not rise above 160/95.

THE REGULATION OF BLOOD PRESSURE

Physiopathology

The following 4 factors are important in regulating the blood pressure: (1) Cardiac output and cardiac force, (2) blood volume, (3) viscosity of the blood, and (4) peripheral resistance. These factors are intimately integrated and controlled by a complex system of humoral and neurogenic reflexes. It will be easier to understand the manner in which hypotensive drugs operate if these four factors in blood-pressure regulation are borne in mind.

In both hypertension and toxaemia there is generalized vasoconstriction of the arteriolar system. This vasoconstriction is in turn responsible for an increase in peripheral resistance; studies on the kidney and on the brain have shown a greatly increased vascular resistance. Burt (1950) in his symposium on haemodynamics in pregnancy, states that peripheral areas such as the skin and the muscles of the extremities are exempt from this vasoconstriction. The increased vascular resistance in the kidney causes a reduction in renal blood flow and in the glomerular filtration rate. Both neurogenic vasoconstrictor impulses and humoral factors are responsible in varying proportions for the arteriolar constriction. In toxaemia it is the humoral factors that predominate in the elevation of vascular resistance. The vasoconstriction has a damaging effect because it results in anoxaemia, retention of waste products, oedema, and necrosis. Autopsy shows ischaemia in most organs and varying degrees of infarcts in the placenta. The principal causes of death are cerebral haemorrhage, cardiac failure, and the accidental haemorrhage and anuria syndrome. Deaths from hyperemesis gravidarum and acute yellow atrophy are very rare. An acute cardiovascular accident overshadows the toxaemia problem. Stated in another way, hypertension is the most resistant feature in toxaemia while the other aspects of the varying toxaemia syndrome have proved much more amenable to control. Corkill (1957) analysed 435 cases of eclampsia occurring in New Zealand between 1950 and 1955: the principal causes of death were massive cerebral haemorrhage (5), sudden cardiac failure from toxic myocarditis (5) and the accidental haemorrhage and anuria syndrome (4).

Rationale for Employing Hypotensive Drugs

In general, the severity of pre-eclampsia is proportional to the degree of hypertension. In the previous paragraph I have quoted several references which show that the vasoconstriction has a damaging effect on tissues. While it seems logical that the vasospasm of toxaemia should be treated, such treatment was frowned upon by many on the grounds that hypertension was a necessary compensation to ensure that blood flows adequately through areas of increased vascular resistance. It was contended that to lower the blood pressure was dangerous because a sufficient pressure was then not available to give the vital organs an adequate supply of blood. Another argument that has been raised is that in reducing the blood pressure one is treating a symptom and not the disease. The fact is that until the 'toxin' responsible for toxaemia of pregnancy has been isolated treatment will have to be symptomatic. Since the chief cause of maternal death is an acute cardiovascular accident, which in turn is the end-result of uncontrolled hypertension, we shall look towards the hypotensive drugs to aid us in controlling this pernicious complication. The mere lowering of blood pressure is not enough; the ideal drug must also improve the circulation of the kidneys, brain and placenta. The alternative to treatment with hypotensive drugs is the time-honoured use of sedatives. Stroganoff, in 1897, advocated the use of morphine, which was and still is used widely. The other sedatives in common use are parenterally administered paraldehyde, barbiturates and magnesium sulphate. McCall *et al.* (1952) studied the effects of heavy sedation on the brain and was disturbed to discover that these agents (especially the intravenously administered barbiturates) depress oxygen metabolism of the brain almost to the same

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degree as eclampsia itself. Others have demonstrated a depressing effect on kidney function and urinary output. Another disadvantage of heavy sedation is the clinical undesirability of prolonged coma and the depressing effect on the foetus. Once the blood pressure is controlled heavy sedation is unnecessary in the vast majority of patients. By controlling the blood pressure it is possible to avoid the eclamptic convulsions and the subjective symptoms of headache, disorientation and blurred vision so commonly present in the hypertensive crises. The avoidance of heavy sedation offers the best chance of survival for the foetus and tends to reduce the incidence of respiratory morbidity in the mother. The comparison between the wide-awake, cooperative patient and the comatose women under heavy sedation who, despite skilled nursing, is liable to develop pulmonary complications, is most striking. As delivery approaches the claims of the foetus may conflict with the requirements of the mother as regards heavy sedation.

The consensus of opinion is that reduction of an elevated blood pressure without reducing the cardiac output and the blood supply to the vital organs is a sound and logical aim. It reduces the cardiac work, and prevents cardio-vascular accidents. As Assali (1954) points out, 'Just as lowering the fever may permit the patient to tolerate the toxic process more successfully, reduction of excessive hypertension may help the patient to tolerate better the ravages of the disease and its noxious effects on the vital organs'.

THE OBSTETRICAL AIM IN TREATING HYPERTENSION OF PREGNANCY

From the obstetrical point of view the aim is to obtain a sufficient degree of maturity of the foetus to render probable a fair chance of survival.

1. *Pre-Eclamptic Toxaemia*

The majority of cases of pre-eclamptic toxaemia respond to bed rest, diet and sedatives. In the minority where this simple treatment is unsuccessful, do the hypotensive drugs help? After the 35th week, when obstetrical viability has been attained, pregnancy can be terminated by surgical induction if conservative medical measures fail to control the hypertension. But if the cervix is 'unripe' the procedure is fraught with grave risks. To 'ripen' the cervix with a Pitocin-drip may take a few days. During this interval can one depend on the hypotensives to control the blood pressure?

Another group of cases which present a great deal of difficulty are those in which the toxaemic manifestations commence before the 35th week. If the toxaemia commences early, the development of the baby is often severely affected owing to deficient nutrition resulting from the associated placental damage. If there is a heavy daily loss of albumen in the urine, intra-uterine death of the foetus is common. The problem in this group is two-fold. In the interests of both mother and foetus Caesarean section is indicated. But because of prematurity one aims at continuing the pregnancy up to the 35th week. The continuance of such pregnancies with a view to gaining further maturity is liable to result only in intra-uterine death, and the prompt decision to terminate them once the 35th week has been reached will often result in saving a baby who otherwise would have been lost. Has the use of hypotensives altered the picture in this group? Can it reduce the Caesarean-section rate and prematurity rate for this group?

2. *Essential Hypertension and Pregnancy*

If the pressure remains within moderate limits and there is no albuminuria, these cases usually terminate normally. The real danger associated with chronic hypertension is the supervention of pregnancy toxaemia, which forthwith changes the prognosis to one of grave danger to the foetus and appreciable risk to the mother. The main aim of the obstetrician is to carry the pregnancy till the 35th week so as to attain foetal maturity before resorting to termination of the pregnancy. If strict bed rest and sedation fails to control the hypertension the judicious administration of a hypotensive may be helpful. In the cases where the basis is essential hypertension the ganglion-blocking agents may be more successful. Once albuminuria occurs there is a very high risk of intra-uterine death of the foetus. A liberal view should be taken of termination by Caesarean section. The fulminating cases in which the blood-pressure readings are excessive (systolic pressure over 200 mm. Hg) may become evident at an early stage, often about the 6th or 7th month. In such cases the mother is exposed to grave danger and the outlook for the foetus is hopeless. In many cases the problem resolves itself by the intra-uterine death of the foetus, which is speedily accompanied by a lessening of the maternal symptoms.

Advantages of Hypotensives over Sedatives

Cerebral Physiology. Recent experimental studies of McCall and Taylor[3] on cerebral blood flow and oxygen metabolism in toxaemia show that barbiturates (especially those administered intravenously) *depress* cerebral blood flow and are likely to prolong the coma of eclampsia. The basis of the Rotunda treatment with intravenous Pentothal drip is to keep the patient unconscious for the 24 hours after the last fit. In their series of cases (Finnerty and Fuchs) no veratrum-treated patient was unconscious for longer than 2 hours. The persistence of coma in eclamptics is a very grave sign; Dieckmann (1952) quotes a maternal death rate of 34-73% of patients. Heavy sedation, besides adding to the complexity of the management of seriously ill patients greatly increases the incidence of respiratory complications.

The hypotensive drug allows the patient to cooperate (*a*) with adequate oral fluid intake and (*b*) by informing the doctor of anything untoward in her symptoms.

Foetus. The heavy sedation is harmful to the foetus, especially as most foetuses are premature, since toxaemia is commonest at 7-8 months.

Pulse. A constant finding in toxaemia, especially in eclampsia, is tachycardia. Dieckmann[5] states that an increase in pulse rate over 120 is associated with a mortality of 39%. In one of our cases we specifically employed Veriloid, in preference to Nepresol, for its bradycardic effect. The patient was an eclamptic with the pulse racing at 170 per minute. She was initially given the Rotunda treatment; after Veriloid therapy the pulse settled at 140 per minute.

Properties of an Ideal Hypotensive Drug

An ideal drug should be (1) rapid in action, (2) prolonged in effect, and (3) harmless to mother and foetus, (4) should have no unpleasant side-effects, (5) should not interfere with

labour, and (6) should have a wide safety margin between toxic and therapeutic dose.

Browne and Veall (1953) using radio-active sodium, showed that the maternal placental blood flow was reduced to one-third in pre-eclampsia and hypertension. It would seem that the ideal drug would be one which would lower the maternal arterial pressure and increase the blood flow through the placenta. A mere fall in the blood pressure without a concomitant dilatation of the spiral arterioles of the placenta might lead to further anoxia of the foetus. A reduced blood supply to the placenta must at a certain stage interfere with its normal functions; an indication which is relatively easy to measure is the transport of oxygen to the foetus (Walker 1954). In a proportion of pre-eclamptics there is a reduced saturation of oxygen in the umbilical vein.

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STEATORRHOEA DUE TO INTESTINAL RESECTION AND STRICTURES

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The resection of large amounts of small bowel is likely to produce evidence of malabsorption. The clinical picture then encountered differs somewhat from that seen in the sprue syndrome[9] and in those conditions where stagnation of intestinal contents (e.g. blind loops, multiple strictures) is responsible for the steatorrhoea.[8, 10] In the case now presented several factors were combined: he suffered from massive resection, a blind loop (thought not to be significant) and intestinal strictures, with the added complication of mucosal ulceration. Partial relief was obtained from removal of the strictured-ulcerated area, and at present the patient's metabolic status presumably reflects the fact that his residual small bowel consists of only 75 cm. of upper jejunum.

CASE REPORT

W.F., a Coloured male then aged 27 years, was first admitted to this hospital on 20 November 1954, complaining of attacks of abdominal pain and vomiting during the previous 5 years, and recent weight loss. Physical examination revealed dextrocardia (subsequent investigations showed that he had total situs inversus). There was moderate gynaecomastia but the testes felt normal. The blood pressure was 130/80 mm. Hg. There were no abnormal findings in the abdomen. The urine was normal and the blood indices were Hb. 15 g.%, ESR 10 mm. in the first hour (Westergren), and WBC 11,000 per c.mm. The Wassermann reaction was negative. Several stools contained occult blood, but a sigmoidoscopy was negative. The urinary 17-ketosteroid excretion was 8·3 mg. in 24 hours.

During an attack of pain the abdomen became distended, with increased bowel sounds; a plain X-ray film at that time showed fluid levels in the small bowel. Laparotomy was performed: a 5-cm.-long constriction was found 15 cm. from the ileo-caecal valve, and 25 cm. of terminal ileum were resected; histology (Dr. J. A. H. Campbell) was that of regional enteritis (Crohn's disease).

The patient did not remain well for long, and he was readmitted on 26 January 1955 with severe abdominal pain. At operation there was localized peritonitis in the ileo-caecal region, loops of ileum being matted together. An unstated length of small bowel was resected, together with the caecum, and an anastomosis was made between the end of the remaining small intestine and the side of the ascending colon. Pathological examination (Dr. M. Sacks) again showed regional enteritis; there were fistulae between the loops of intestine.

Since this operation he has suffered from diarrhoea. At first his bowels acted up to 15 times a day, but after 3 months they became less active. The stools are pale, bulky and offensive. He entered hospital again on 21 May 1957, because the diarrhoea had become worse during the preceding 9 months and because he had developed abdominal cramps and distension and dyspnoea on effort. He weighed 96 lb. and was very pale. His fingers were slightly clubbed. There was a grade-3 blowing systolic murmur, maximal at the apex of the heart. The blood pressure was 130/80 mm. Hg. The gynaecomastia was unchanged and the testes were normal. The abdomen was distended; the liver edge could be felt 5 cm. below the left costal margin. The urine was normal. The blood indices were Hb. 5 g.%, VPC 20%, ESR 1 mm. and WBC 5,000 per c.mm.; the peripheral blood smear was hypochromic. Some of the relevant investigations are listed in Table I. The blood urea and electrolytes were normal. On electrophoresis, the serum-protein pattern was normal. Apart from the evidence of dextrocardia, the electrocardiogram was within normal limits. Uropepsin excretion was 4 units per hour and he secreted 10·6 mEq. of free HCl in 1 hour in an augmented histamine test (both normal). Bacteriological examination of the stools was negative, but they contained occult blood. The bone marrow was considered to be basically normoblastic, with evidence of iron deficiency, but several cells resembling megaloblasts were seen. A liver biopsy was reported (Dr. W. B. Becker) as showing marked fatty change. Barium studies confirmed the situs inversus; both enema (Fig. 1) and meal (Fig. 2) showed a narrowed segment of small bowel near the anastomosis, and the meal showed flocculation.

TABLE I

	Period I	Period II	Period III
Serum			
Proteins (g.%)			
Albumin	3·9	4·3	3·9
Globulin	1·6	2·5	2·8
Cholesterol (mg.%)	109	250	101
Calcium (mg.%)	7·4	11·5	9·6
Phosphorus (mg.%)	4·2	4·2	
Urine			
Calcium (mg./24 hours)	63	105	52
17-ketosteroids (mg./24 hours)	3·6		6·1
Tests of Absorption			
Xylose (g. excreted/5 hours)	3·6		2·7
Fat (%) (3-day balance, 70 g./day)	65	33	56
Glucose tolerance curve	Flat	Flat	Flat

Periods
I: Pre-operative, May 1957.
II: Soon after operation, August.
III: 4 months after operation, November.

Fig. 1. Barium enema.

The anaemia did not respond to the parenteral administration of vitamin B12 and folic acid. He received a high-calorie diet, vitamin supplements, oral chlortetracycline and blood transfusions, and his weight rose to 109 lb. At this stage the cardiac murmur was no longer audible.

On 15 July 1957 Dr. D. J. du Plessis performed a laparotomy. The small bowel was found to be only 90 cm. long, and to be dilated and hypertrophied. Proximal to the entero-colostomy site was a blind loop of ascending colon, 15 cm. long. In the jejunum were 2 ring-like narrowings 5 cm. apart, the distal one being 4 cm. from the anastomosis. The distal 15 cm. of small bowel were resected, with the blind pouch of ascending colon, and an end-to-end jejuno-colostomy was made; approximately 75 cm. of small intestine were left behind.

The mucosa at the site of the strictures was ulcerated. Histologically, again, the picture was that of regional enteritis (Dr. C. J. Uys). He made a good recovery from the operation. The bowel actions have decreased in number from 6-7 a day soon after the operation to 3-4, 4 months later. When re-assessed in November 1957 his weight was maintained at 110 lb., and he was neither dyspnoeic nor anaemic (Hb. 15 g.%, smear normochromic). The blood pressure was 120/80 mm. Hg. He had not suffered from a recurrence of the abdominal pain. The electrolytes and blood urea were normal. A barium meal with the usual type of suspension again showed clumping; with a non-flocculating medium the mucosal pattern was normal and the jejunum was slightly dilated. Other data are recorded in Table I.

DISCUSSION

The history at the time of the patient's admission in May 1957 suggested that he had steatorrhoea, and this was confirmed by the fat balance. The biochemical tests (flat glucose tolerance curve, low xylose absorption) and the flocculation-pattern on barium meal indicated an enterogenous cause.[9] The barium studies also showed a narrowing proximal to the entero-colonic anastomosis; as the previous operations had been for regional enteritis, a recurrence was thought to have produced strictures. In view of the positive tests for occult blood in the faeces, indicating mucosal ulceration, active enteritis was diagnosed. The abdominal pain was thought to be due to partial intestinal obstruction. Laparotomy confirmed these impressions.

Multiple intestinal strictures cause steatorrhoea by producing a 'stagnation syndrome'.[6, 9] Megaloblastic anaemia is often found; in the present case the bone marrow was probably normoblastic and the anaemia did not improve on treatment with vitamin B12 and folic acid. It was obviously due largely to blood loss, but malabsorption may have contributed to the picture.

The post-operative course has shown conclusively that the strictures were not the principal cause of the steatorrhoea; in fact, 4 months after the operation the fat absorption was lower than it had been prior to surgery. Obviously, the extensive intestinal resection must be incriminated. Had his intestinal length been adequate, excision of the strictured area would have caused the fat absorption to return to normal, as occurs after restoration of proper bowel continuity in the blind-loop syndrome.[10]

What did surgery achieve in this patient? (1) The source of blood loss has been removed. (2) He no longer suffers from intestinal obstruction. (3) The strictures can no longer interfere with absorption (assuming that they might have done so).

The radiological sign of clumping of barium has been said to occur only in those cases of steatorrhoea which are due to

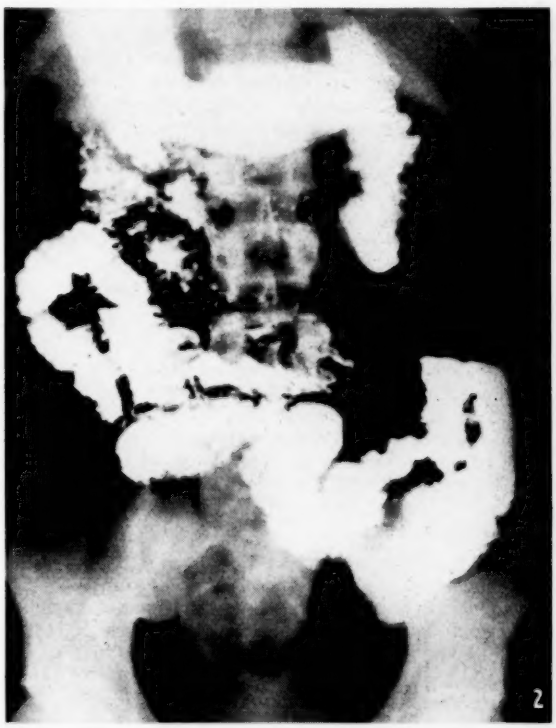

Fig. 2. Barium meal.

sprue, Whipple's disease and intestinal lymphoma;[12] it is thought to be due to flocculation of the barium by mucus secreted in response to the presence of unabsorbed short-chain fatty acids. Gardner[6] did not find this sign in blind-loop steatorrhoea, and one would therefore not expect it in the analogous condition of multiple strictures. Changes in the mucosal pattern with non-flocculating barium are probably of greater significance in the radiographic investigation of malabsorption.

Regional enteritis has been thought to cause steatorrhoea on the basis of extensive bowel disease, but it is likely that localized strictures are more often to blame. Support for this suggestion is obtained from a study by Ensrud and Sauer,[5] who divided their 38 medically-treated cases of regional enteritis into 3 groups, according to the amount of bowel involved. Of the 11 patients in whom less than 12 inches of the bowel was affected, 4 had a malabsorption syndrome, whereas of the 19 in whom more than 36 inches was diseased, or in whom there were skip lesions or internal fistulae, steatorrhoea was noted in 5. Only 1 of the 8 cases in the intermediate group suffered from malabsorption.

Jackson[7] states emphatically that metabolic disturbances are rare in patients in whom less than 2/3rds of the small bowel has been resected, provided that the remaining segment is healthy. In the present patient the possibility of recurrent enteritis must always be a hazard, still further to deplete his limited absorptive capacity. His residual bowel length approaches the danger mark; with less than 18 inches, the prospect is grim.[7] Pietz[13] notes that patients retaining a segment of ileum appear to do better than those having an equivalent length of jejunum, and suggests that the ileo-caecal valve may be the important factor. An alternative explanation may be deduced from the work of Benson *et al.* in rats;[1] they found that olive oil was preferentially absorbed from the third quarter of the small intestine. However, a recent study of normal humans by Borgström *et al.*[3] contradicts this thesis; the intestinal contents were sampled at various levels, and it appears that fat, protein and carbohydrate are almost completely absorbed within 100 cm. of the duodeno-jejunal flexure. In spite of this, it is possible that lower segments of small bowel may be able to take over some of these functions—this aspect remains to be studied.

According to Booth and Mollin[2] vitamin B12 is absorbed from the ileum, and not from the proximal small intestine. This is difficult to reconcile with Jackson's finding that megaloblastic anaemia does not occur after experimental or clinical bowel resection, whether the ileum has been removed or not.[7] Chronic blood loss was thought to be the principal cause of the anaemia in the present patient, who had not responded to parenteral vitamin B12 and folic acid, but the strictures and the absence of the ileum may have been aggravating factors.

Linder *et al.*[11] have defined the effects of massive resection of the small intestine as *undernutrition with special adaptation*. At this stage the present patient shows little secondary evidence of depletion and is in a better nutritional state than before his latest operation. When he presented in May 1957 he had hypocalcaemia, hypoproteinaemia and a fatty liver, but there were no signs of vitamin-B deficiency.

In contrast with other cases of massive resection showing the undernutrition-adaptation syndrome, the patient under discussion has a normal blood pressure, has not lost libido, does not crave salt, has a normal serum-potassium level, shows no evidence of psychological deterioration, and is able to maintain his weight. His urinary 17-ketosteroid excretion is higher than it was before the strictures were resected, but slightly lower than it had been when he first attended in 1954.

In spite of the severe steatorrhoea he shows signs of improvement—his fat absorption, at its nadir soon after the latest operation, has risen to 56%. His post-operative progress may be due to continuing adaptation of the remaining intestine; this must also have occurred after the second operation, when the intense diarrhoea settled down after a few months. As in 'Toni', the case of massive resection reported by Jackson and his associates,[8, 11] the remaining jejunum was dilated and hypertrophied, but this may have been partly due to chronic intestinal obstruction. The blind loop probably had no metabolic significance as it presumably was able to empty itself with peristalsis[4] and consisted entirely of colon. It is unfortunate that the length of the bowel removed at the second operation was not recorded; one gains the impression that the resection may have been somewhat generous. There is nothing to suggest that his small intestine was abnormally short before the operations. Although his ultimate prognosis is poor, he is not in immediate danger of death from inanition if the enteritis does not attack the remaining bowel.

The gynaecomastia and situs inversus do not appear to have any relationship to the bowel condition or its effects.

SUMMARY

A patient who had previously undergone extensive bowel resections for regional enteritis is discussed. He was studied while suffering from inflammatory strictures in his remaining 90 cm. of jejunum, and again after the diseased area had been excised. He showed the features of enterogenous steatorrhoea, anaemia due to blood loss, and partial intestinal obstruction. Resection of the affected bowel, in which the pathological process was recurrent regional enteritis, cured the latter two complaints. It was not possible to evaluate the role the strictures played in the production of malabsorption.

I wish to thank Dr. Louis Mirvish for permission to report this case. Dr. W. P. U. Jackson kindly allowed me to read his contribution to Modern Trends in Gastro-enterology[7] in proof. The cooperation of Dr. Velva Schrire and Dr. D. J. du Plessis is greatly appreciated. Mr. B. Todt made the reproductions of the X-ray plates.

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NEUROTIC CHOICE OF A MARRIAGE PARTNER*

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The concept of a neurotic choice of mate connotes a marital selection which in itself may interfere with normal relationships, or may make them so difficult that the displeasure exceeds the pleasure derived from them.

The purpose of this paper is not directed at proving that all neurotically motivated marriages are unprofitable, nor is it directed at an exhaustive survey of the various categories of neurotic motivation, nor are whirlwind marriages and frivolous contracts considered. Furthermore, there is no reference to neurotically motivated marriages where the motive is conscious or partly conscious, e.g. flight from homosexuality, flight from intolerable home circumstances, gallops towards material security, and others.

The cases illustrated deal with the marriages of intelligent adults whose conscious motives were of the highest order, and who seriously considered the steps they were taking. The deeper unrecognized motives, however, had their origins in the early relationships of one or both partners, and the marriages when put to the test were found wanting. Such marriages not infrequently present themselves in the consulting rooms behind the guise of unrelated symptoms. The clinician's diagnostic and therapeutic ability, his insight into human behaviour and his most personal moral code are diversely challenged by many of these problems.

The purpose of this paper is to draw attention to the frequency of these cases and the challenges they present, and to evoke a greater medical responsibility towards marriage.

The four cases briefly reported presented major symptoms other than marital distress. The marital unhappiness uncovered in each is traceable to neurotic attitudes which played an important part in the choice of a mate.

CASE RECORDS

The first case concerns the marriage of an aggressive woman who is determined to hold the upper hand in her relationships with any males, but who nevertheless finds the position of superiority she strives for intolerable.

The patient, aged 32 years, presented the symptoms of tension, depression and threatened suicide, present intermittently from the time of marriage 5 years previously and severe for some months.

She was born in the USA, the second of 4 siblings. Her father was a research chemist who preferred her younger brother. Her sisters were more attractive and successful socially than she. In school and nursing, the profession she chose, she sought and achieved high marks. When 17 years old she was alarmed by her own fondness for an older woman, the leader of a girl's movement. This lasted some months but was not acted out. Lack of social success concerned her. In her early twenties she was seduced by relative strangers on half a dozen occasions, achieving no satisfaction.

At the age of 27, she met and married her husband, who was of the same age. He was a travelled, intelligent, passive person, who treated her with absolute respect. After marriage it became clear that this was due to his shyness, anxiety and inexperience. Marriage was followed within the first 3 years by the birth of twin boys and then a third son. Economic and physical hardship ensued, because of an overseas training programme. Her husband, inexperienced sexually, had occasional ejaculatio praecox. She complained of his inability to 'conquer her' sexually, and remained sexually unsatisfied. She reproached him for his submissiveness, but reacted with severe depression whenever he became critical of her, or aggressive. She challenged his ability at the commencement of love-making and belittled him when he failed. Consciously she craved acceptance and affection from her husband, but motivated by an intense resentment against her father, brother, and all males, she persisted in humiliating him to the point of exasperated helplessness.

The second concerns the reciprocal choice of an emotionally detached male and a dependent female craving affection. The male, basically insecure, uses emotional detachment as a defence. This provides him with a façade of strength which attracts his dependent wife. He, on the other hand, regards her vivacity, particularly as she has a vocation, as independence, and thinks she will make no demands on him either for emotional support or for open affection. Early in the marriage the first difficulties arise through the growing disillusionment of both parties in the sphere of genital satisfaction. Later the marital difficulties are precipitated by events outside of the direct relationship.

The patient, aged 34, obsessional and perfectionistic, extremely obese yet not unattractive, complained of irritability, tearfulness and depression and preoccupation with suicide, intermittently for a few years.

She was the second of 2 siblings, having a brother 7 years older than herself. Her father, who had favoured her openly, died when she was 19. Her progress through school and in her secretarial career indicated her high capabilities. She had been obese from her early teens.

Her marriage when 18 years old to a man aged 30 had initially been fairly happy, despite her disappointment at the lack of physical affection. Two daughters resulted from the marriage—one 14 years old and one 10. The elder child, by the age of 5, was obviously below par intellectually.

Considerable organization was then undertaken by the patient to assist the child, whose IQ was 72, to overcome her handicap, and at the age of 14 years she had attained a fair standard in some Standard V subjects at a convent school. The high level of orderliness and tidiness in the home had been in no way reduced to accommodate the backward girl. The patient's disappointment was ntense when the child's best was just not good enough.

Her husband had refused to discuss his daughter's backwardness with his wife. The more intense her emotional reactions to the problem, the greater his withdrawal. He was prepared to discuss it 'logically and rationally' only, and her tears sent him into a state of sullen moodiness which would sometimes last for 2 weeks. He was described by her as a good and faithful husband who was, however, failing to support her in the major crisis of her life. His rejection of her extended into an already only partly satisfactory sexual relationship, to the point where she described his approach as thoroughly selfish. They appeared to be failing each other hopelessly.

The third concerns the choice of a highly intelligent professional woman, who has resented the domination of a driving intellectual professional mother throughout her years of development. She marries a noted handsome athlete of mediocre intellectual endowment in the face of enormous opposition from her mother. She sincerely believes that her mother's philosophy is wrong and unrewarding. Her deeper unrecognized need is to triumph over mother and intellectual achievement which mother stands for.

Despite the neurotic need motivating the marriage, all might have been fairly well had fate not played the unkind trick of reactivating her original conflict with her mother by presenting her with a mentally backward child. The presenting symptom is the child's backwardness.

This highly intelligent, grossly immature woman, aged 30 years, complained of her elder child's difficulties with school-work, and

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insisted that something be done to improve her. She considered her daughter's retardation an unbearable disgrace.

She was the eldest of 3 siblings, having 2 brilliant younger brothers. Her mother was a professional woman, and her father a highly intelligent self-made man. At school and university, despite the achievement of top marks, she was constantly reminded by her mother, for whom she had little affection, that her cousins and brothers could do better.

Having completed her university degree, she was married at 19 to the good-looking athlete, who was 3 years her senior, against strong family opposition based on the belief that he was intellectually inferior to her. Despite their minimal common interests and totally different avenues of endeavour, her determination to marry him increased proportionally to her mother's devaluation of his social status and intelligence. Two daughters resulted from the marriage, the elder aged 9 years with an IQ of 78, and the younger aged 4 years, who was very bright.

The family supposition was deeply rooted that the inferior intellect could only have been introduced by the father's genes, and the maternal grandmother's triumphant reproaches were strongly reminiscent of her attitude towards intellectual achievement during the patient's years of school and college. The anxiety which this precipitated had led to the patient's quarrelling with her daughter's school principals for not taking enough interest in the child's education, and had brought her to psychiatry.

By her marriage, she had hoped to triumph over the aspects of her mother's philosophy, which had punished her so sorely during her years of growing up. The idols she had smashed by means of her marriage, had returned to life with a renewed vigour and fearsome vengeance.

The fourth concerns the choice of a good-looking young man who has stammered from his early childhood and is challenged by the cold aloofness of an attractive woman. He marries with the conscious belief that he has met the first woman whom he can respect; she is the first to withstand his sexual advances. His deeper motive was to meet the challenge she presented, viz. to win her sexually. He finds that she is as unconquerable after marriage as she had been before.

This impeccably dressed, good-looking young man, aged 28, was referred to psychiatry by a speech therapist who attributed the negligible success of 3 months of speech therapy to his anxiety and tension. His complaint was a gross aggravation of his stammer since his marriage.

He was the second of 3 siblings, having a domineering mother and a weak unsuccessful father. He was particularly close in an immature dependent way to his mother, who constantly belittled her husband in her family's presence. From his early childhood he fought and almost mastered a stammer which his family and mother especially regarded as a sign of inferiority. As a means of compensation he became keenly competitive in his relationships with his school-fellows, playing all games to win. This attitude followed him through childhood and adolescence, and on entering adulthood the collection of sexual victories became an absorbing and successful pastime.

At the age of 24 he was challenged for the first time by the cold aloofness of the girl who withstood all his sexual advances. After 2 years of courtship he persuaded her to marry him. Her continued refusal after marriage to submit readily to his advances led to a bitter struggle, crowned by frustration and resentment. As the tension in the marriage mounted, so his stammer worsened.

These 4 cases clearly illustrate the tragic discrepancy between conscious needs and unconscious goals. This discrepancy constitutes the major source of the patients' distress and emerges immediately after marriage in the first and fourth cases, whereas in the other two it becomes manifest only after other factors supervene.

It is clear, in the evaluation of the psychodynamics of these people, that their early relationships with parents, siblings and friends gave rise to attitudes which perpetuate conflict and which, by being carried through adolescence into adulthood, influenced their choice of a marriage partner. Distress resulted equally when the marriage actually fulfilled the neurotic requirements or when it failed by not meeting the expectations.

Depending on the maturity that therapy provides, partners, instead of tearing each other to pieces, may agree in all friendliness that each had made an error of judgment in good faith, and decide to separate. On the other hand, maturity and insight sometimes lead to a greater understanding between the partners of their respective needs, and on this may be constructed a happier and more successful marriage. The decision ultimately must lie with them.

It may be easy for the doctor to advise divorce or extramarital sexual adventures, but I submit that these recommendations, even when given in good faith, are ill-advised. In contrast with this god-like demeanour of the one clinician is the diametrically opposed attitude of the other, who believes his obligation to the marriage is exonerated by limiting his concern to his patient only. A greater understanding and acceptance of his responsibility to marriage by the family doctor and psychiatrist, would contribute immeasurably towards a greater knowledge of the problem, and towards a greater degree of marital adjustment.

ANURIA FOLLOWING PENICILLIN ADMINISTRATION

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Untoward reactions to penicillin have given rise to increasing concern over recent years. Anuria occurring as a manifestation of penicillin sensitivity has been described on a few occasions. Langdon described exfoliative dermatitis and renal shut-down occurring in a 53-year-old male after an injection of penicillin.[1] Swann and Merrill[2] recorded 5 days of anuria in a 48-year-old male which they ascribed to penicillin sensitivity. Carré and Squire[3] described the case of a girl of 8 months who 1½ hours after an injection of crystalline penicillin for earache, vomited, appeared shocked, and passed excessive quantities of urine: after a second injection she developed an evanescent erythematous rash and anuria.

In the following case of anuria in infancy it is suggested that the anuria was a sensitivity reaction to penicillin.

CASE REPORT

A European boy of 13 months was admitted to Groote Schuur Hospital on 3 February 1955 because of failure to pass urine. He had been well until 30 January, when he developed symptoms of an upper-respiratory-tract infection. On 1 February he was given an injection of procaine penicillin by his private practitioner. Soon after the injection he became restless, cried much, and vomited

persistently. He failed to pass urine after the morning of 2 February and at the time of admission had been anuric for 30 hours. He was constipated and had become increasingly drowsy over the 24 hours preceding admission.

He was a well nourished child weighing 22 lb., apyrexial, and showing no clinical evidence of dehydration. The bladder was not palpable and the genitalia were normal. The systolic blood pressure was 99 mm. Hg. The ocular fundi were normal.

Special Investigations. Haemoglobin 10·6 g.%. White blood count 7,350 per c.mm. Blood urea 200 mg.%. Serum: Na 124·3 mEq./per l., Cl 88 mEq./per l. Co_2-combining power 26 vols.%. Wassermann reaction negative.

Course and Treatment

The patient was fed on 5% dextrose solution. During the night he started to vomit and passed dark, liquid stools. The vomiting became more persistent over the course of the next day, despite feeding by gastric drip and an injection of 12½ mg. of Largactil. His condition deteriorated and on the evening of 4 February he developed generalized convulsions. The systolic blood pressure at this stage was 110. The convulsions were controlled by intramuscular injections of 2 ml. of paraldehyde and ½ gr. of sodium gardenal, and lumbar puncture. The cerebrospinal fluid was normal.

On the morning of 5 February the child passed approximately 2 ml. of urine, containing albumin but no other abnormal constituents. On 5 February, when anuria, apart from this, had lasted 84 hours the stomach was washed out, with the return of much mucus and some blood. A modified 'Borst' regime was instituted: a mixture consisting of 40 g. of salt-free butter, 160 g. of glucose and 240 ml. of water per 24 hours was given by stomach tube, and 24-hour urinary collections were started. At the end of the first 24 hours on this regime the mixture had been well retained; anuria was still present, but the patient's general condition had improved and the convulsions had not recurred.

The patient started to pass urine during the second day of the regime (7 February); 55 ml. was passed, after an anuric phase of 96 hours. This urine contained a trace of albumin and scanty hyaline and epithelial casts. 60 ml. of M/6 sodium lactate was added to the gastric drip to compensate for the volume of urine passed and to combat the tendency to acidosis.

The blood chemistry after the first diuresis was as follows: Na 131·8 mEq. per l., Cl 74·0 mEq. per l., K 2·55 mEq. per l., CO_2-combining power 39 vols.%, blood urea 279 mg.%. To correct the low serum Na, Cl and K 1 g. of NaCl and 0·5 g. of KCl were added to the gastric drip. Over the next few days the urinary volume increased progressively. The intake was accordingly increased. When vomiting occurred its volume was assessed and an equal quantity of the 'Borst mixture' was added to the gastric drip. In view of repeatedly low serum Na, K and Cl electrolytes were added to the feeds, as shown in Table I. By 13 February the patient was taking cow's milk from a bottle and a few days later he was on a mixed diet.

On 17 February a generalized urticarial eruption developed, which responded to Benadryl.

The blood-urea level gradually fell until by 23 February it had reached 34 mg.%. The 24-hour urinary volume at this stage was 630 ml. and the specific gravity was 1·012. A subcutaneous pyelogram revealed adequate concentration and excretion of Uriodone.

Investigation of Sensitivity. Dilute solutions of penicillin in saline were used for intradermal injections. Areas of redness 1·5 cm. in diameter but without induration developed in response to a solution containing 2·5 units in ·01 ml. No redness was obtained in a control. Solutions of procaine hydrochloride did not produce any reaction. Attempts to demonstrate the presence of specific antibodies by the Prausnitz-Küstner technique were unsuccessful.

Subsequent progress. One month after discharge the patient developed an attack of gastro-enteritis during which he became dehydrated so as to require a subcutaneous infusion, but the urinary output remained satisfactory.

Management

As indicated above, a conservative dietary regime was employed, a high-calorie, high-carbohydrate, protein-free and electrolyte-free diet being given, with restricted fluids.[4,5] The caloric requirement was assessed on the basis of 100 calories per kg. per day. This was supplied by means of a mixture of salt-free butter and glucose in the proportion of 1 to 4 in a volume of water equivalent to the estimated insensible fluid loss. The insensible fluid loss was calculated on the basis of 1 g. per kg. per hour.[6] This estimate may well prove too conservative, especially when diarrhoea occurs. With the onset of diuresis the volume of the fluid intake was increased by an amount equal to the urinary loss. The fluid intake was further increased by amounts approximately equal to the diarrhoeal fluid loss.

Biochemical analysis of the blood was carried out frequently and electrolytes added as indicated by the results, once diuresis had started. Hypokalaemia developed despite protracted anuria. This was probably due to potassium being lost in the diarrhoeal stools and was corrected by the addition of Darrow's solution and KCl. When hyponatraemia occurred additional NaCl was prescribed. M/6 sodium lactate was given when acidosis was marked. When vomiting occurred its volume was assessed and corresponding additional quantities of the 'Borst' mixture were given.

DISCUSSION

This case showed several of the features which characterize acute tubular necrosis. Although vomiting was frequent there was never any clinical evidence of dehydration as a cause of the anuria; nevertheless the rapid development of high blood-urea levels suggested that some degree of dehydration was in fact present. Despite an adequate fluid intake after

TABLE I. SHOWING THE MAIN BIOCHEMICAL FINDINGS AND THE TREATMENT

	Serum					*Urine*					*Intake*	
Feb.	*Na*	*K*	*Cl*	*Co_2 combining power vols.%*	*Urea mg.%*	*Na*	*K*	*Cl*	*Vol. ml.*	*S.G.*	*Vol. ml.*	*Composition*
	mEq. l.					*mEq. l.*						
3	124	4·4	88	26	200						*ad lib*	5% dextrose.
5	110	4·1	72	23	205						213	213 ml. 'Borst'.
7	132	2·6	74	39	279	28·8	36·3	38·5	55	1·010	294	234 ml. 'Borst' + 60 ml. M/6 Na lactate.
8	130	3·1	78	40	264	50	31·7	45·9	213	1·040	308	308 ml. 'Borst' + 1 g. Na Cl + 0·5 g. KCl.
9						40·2	24·7	39·3	342		470	350 ml. 'Borst' + 120 ml. Darrow's solution.
10	146	2·5	91·1		214	35·3	21·1		305	1·028	720	480 ml. 'Borst' + 240 ml. ¼ strength Darrow's.
11									366	1·018	900	450 ml. milk + 150 ml. Darrow's solution + 300 ml. water.
12									404	1·023	900	690 ml. milk + 210 ml. water + 1 g. KCl.
13									216	1·041	900	Milk.
14					148				221	1·039		Mixed diet.
23					34				630	1·012		Mixed diet.

The serum levels of sodium, chloride and potassium fell during the course of the illness. The volume and electrolyte content of the stools were not estimated; consequently accurate replacements could not be made.

admission there was still no passage of urine. The hypoelectrolytaemia was probably due to loss of electrolytes in the vomitus and in diarrhoeal stools. While the serum levels of sodium and chloride were low, the urine in the early diuretic phase contained considerable quantities of these ions, a finding in keeping with the diagnosis of acute tubular necrosis.[7]

Acute nephritis may cause anuria. In this case there may have been an antecedent throat infection, but the renal shut-down followed too rapidly for acute nephritis unless there had been a pre-existing renal lesion. The subsequent normal renal function argues against pre-existing renal disease.

Conclusive evidence that the anuria was due to penicillin sensitivity is lacking, but the development of vomiting and restlessness soon after the injection is highly suggestive. The positive response to the intradermal injection of a dilute solution of penicillin is an interesting feature. The development of urticaria 16 days after the injection of penicillin may have been a delayed sensitivity reaction. There was no story of previous penicillin administration, but fatal reactions are reported to have followed the first injection. Sensitization may also result from a previous fungus infection. The similarity between this patient and the case described by Carré and Squire[3] is very striking.

It is suggested that vascular spasm resulted from sensitivity to penicillin. This in turn led to renal ischaemia and acute tubular necrosis. Vomiting, with the later development of diarrhoea, may have been contributory factors in the production of this syndrome.

SUMMARY

A case of anuria occurring in a 13-month-old boy after an injection of penicillin is described.

A conservative dietary regime was employed, with a high-calorie, high-carbohydrate, protein-free and electrolyte-free diet and restricted fluids.

It is suggested that the anuria was a sensitivity reaction to penicillin.

I am grateful to Dr. J. G. Burger, Medical Superintendent, and Prof. F. J. Ford, head of the Department of Paediatrics, Groote Schuur Hospital, for permission to publish this case.

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THE LIFE AND WORK OF WILLIAM HARVEY*

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During 1957, my year of office, the death of William Harvey was commemmorated throughout the civilized world. It is therefore appropriate that I should tonight recall the life and work of this outstanding colleague.

William Harvey was born in 1578 to young and happily mated parents in the 20th year of Elizabeth's reign, with Drake in the 'Pelican', 3½ months at sea on the first English voyage round the world; Hawkins at work, not wholly to his own disadvantage, upon the Navy that was to discomfit Spain; Gilbert at Limehouse brooding over his plan for future overseas colonies; Frobisher on the verge of sailing from Harwich upon the third of his Arctic explorations; Sydney at Court, beloved of the Queen and already encouraging, perhaps by his friendship, Spenser's muse 'out of the floor to sing his sweet delight in lowly lays'; Bacon reading for the Bar, but for the moment in France; Shakespeare and Marlowe boys of 14—and something of what that stood for can hardly have failed to find its way across the Harveys' threshold.

Dr. N. A. Rossiter

Harvey received his early education at King's School, Canterbury, and then obtained a scholarship which took him to Caius College, Cambridge, for 6 years. The scholarship was founded in 1571 by Matthew Parker, Archbishop of Canterbury, and one of the interesting conditions was that at the University the scholar should be educated first in subjects that pertained to medicine, and then in subjects which actually constituted medicine itself. Such a regulation for a medical scholarship was very remarkable, and unique at that time.

In Harvey's 'Prelectiones' there are references to the anatomy of no less than 80 animals, and his book on generation is a mine of information on natural history. Many references are made to Harvey's intense interest in natural history. This intense lifelong devotion to this science is an inborn quality, and it is by no means unlikely that in Harvey's schoolboy days it was this predilection that justified the authorities in nominating him to what was practically a medical scholarship.

Harvey's first term at Cambridge began in May 1593, when he was admitted to the scholars' table at Caius College. Although Caius College had a medical reputation there is rather meagre evidence of systematic instruction in medical subjects. Dr. Caius had obtained a licence for the dissection of two bodies annually in the winter, and for their decent burial. The church registers give evidence that bodies, chiefly of prisoners, were dissected, and demonstrations were given.

The Exiit Book throws some interesting information on Harvey's time at Cambridge. During the first 3 years there were no absences beyond one month's annual holiday. During the next 3 years the Book shows repeated absences, and Harvey was away from college not less than 16 months. We know that Harvey, like Sydenham, suffered severely from gout at various times, but there is no certain explanation of his absences. He took his Arts degree in 1597. Following in the footsteps of Linacre and Caius and of other Fellows of his College, Harvey went in 1600 to Padua, where the standard of luxury was scarcely high. The food was bad, and the windows of the students' quarters consisted for the most part of sheets of linen. Artificial light was a rare amenity, and there was very little amusement for leisure hours.

* Valedictory Presidential Address.

Before we follow Harvey's further career, let us consider for a moment the intellectual atmosphere of Europe when he began his course at Padua. There had been a great revival of classical learning, followed not only by the Reformation and the consolidation of the Protestant states, but by the counter-reformation of the old Catholic countries. And there was a great ferment of free enquiry on philosophy, on the principles of government, on the polity of nations, the rights of the humbler folk, and the conduct of life. The creation and dissemination also of the greatest English literature was then in progress. Tolerant and open-minded Britain welcomed to permanent posts many learned men from other lands. Continental travel attracted many Englishmen who sought illumination in those days far more in Italy than in Germany, or even in France.

Galileo had been lecturing 6 years at Padua before Harvey went there. Robert Boyle was not born till 1627, and the work of Torricelli and of Pascal was still to come. The Royal Society held a few informal meetings in 1645, but did not receive its charter till 1662. Thus, although at the end of Harvey's life physics had made great advances, in 1600 it was by no means a popular science. The chemistry of that time was dominated by unverifiable hypotheses which were too vague and nebulous to suit Harvey.

Of the biological sciences the subject which not only engaged the energies of some of the greatest men of that generation, but also attracted the interest of educated people, was human anatomy. Italy was the Mecca of anatomical students. At Padua itself there was a great tradition, for Vesalius, Falliopus and Fabricus formed a brilliant succession of teachers. Indeed it was in anatomy and surgery, rather than medicine, that the progress of the age was being principally reflected. Harvey found himself in the van of all that was progressive in his chosen career.

Charles I took the greatest interest in anatomy and in the numerous demonstrations, not only on the circulation of the blood, but on embryology, which Harvey gave him from time to time.

But it is important to note that although the great Italian anatomists had made enormous strides in the study of structure, their knowledge of function was lamentably small; physiology had not kept pace with anatomy. It has indeed been stated that physiology had not advanced since the time of Galen, and doubtless its inadequate progress was partly the consequence of the prevailing ignorance of physics and chemistry.

What Harvey's exact attainment was at this time in anatomy and the technique of dissection we have no means of knowing, but we have reason to believe that even then he was by no means ill-equipped with ancient learning. We know that he was a keen and accurate observer and an enthusiastic naturalist, and that he had a mind reflective on the causes and relations of things, fertile in recognizing resemblances and, above all, ready in making working hypotheses and devising experiments which would verify those hypotheses.

From the time when Vesalius described the valves of the heart, the Italian anatomists had been keenly exercised on the valves in various parts of the vascular system; Fabricius' discovery of the valves of the veins was made as far back as 1574. But the scientific use of the imagination seems to have been dormant with these clever observers, and they were inept in explaining the mechanism of the valves.

Sir William Osler suggests that it was reflection on the mechanism of the valves of the veins which at this early stage led his mind in the direction of his own great discovery, and there is a remark of Harvey's friend Robert Boyle which is not inconsistent with that suggestion. Boyle says that Harvey's observations on 'contrivances similar to the valves used in hydraulic engines for preventing counter-currents led to his discovery of the circulation of the blood'. It is certain from the terms of his diploma that Harvey enormously impressed his teachers and examiners with his knowledge and ability.

Harvey returned home in 1602 and, after his incorporation as Doctor of Medicine at Cambridge, he presented himself to the College of Physicians as a candidate, which is equivalent to the present designation of Member. He was elected to the Fellowship in 1607, being then 29. Harvey then became a physician at St. Bartholomew's Hospital. The relations between the Governors and Harvey were creditable to both sides. In 1633 Harvey submitted revised hospital regulations, and these were adopted; they show that Harvey was a strong conservative and that he stoutly maintained the then existing supremacy of physicians over surgeons. The surgeon was not only obliged to consult the physician in all difficult cases and whenever inward physic was required, but he was not to be allowed to perform any major operation without the approbation and the direction of the physician. The surgeon was forbidden to give inward physic and had to attend the physician in his weekly consultations in the Great Hall, to which the patients were brought and where both matron and apothecary attended. In those days the surgeons had to visit their patients in the wards, and the physician as a rule only went to the wards when his patients could not be brought to the Hall, where most of the medical work was done.

Harvey was connected with St. Bartholomew's for 36 years. His post of Court physician, first to James I and subsequently to Charles I, led to his having a considerable clientèle amongst the well-to-do. We know that some of the most eminent men of time were his patients. It is said that his practice declined sharply after the publication of his 'De Motu Cordis'.

Harvey's Court duties were by no means strictly limited to medicine. As physician to the Court he was instructed to take two long and important continental journeys. In 1630 he accompanied the young Duke of Lennox on a rather prolonged tour, during which he several times dodged the plague and was greatly impressed with the impoverishment of the countries through which they travelled. In 1636, he accompanied the Earl of Arundel, the Lord High Marshall, on an embassy to Vienna and Ratisbon, which was mainly concerned with efforts to secure the restoration of the King's nephew, the young Prince Palatine, to his proper status.

It was during a visit to his friend Lord Feilding that he was detained at the border for fear that he might be carrying the plague. Harvey refused to go to the lazaretto and elected to stay in the open fields. Detained, Harvey writes to Lord Feilding: 'I am fallen into the hands of a base and evil people and while they delight to exercise their tyranny I am to lie in the open fields for God knows how long, and I fear it will do hurt to my health'. As a result of this letter Harvey was offered a roof and a bed, but this he declined because, as he said, 'such offers were unseasonable, like physic when a man was dead'.

A few days later he writes: 'Their manners and cruelty have been shameful, and I now have a sciatica that maketh me lame. I have never longed for anything so much as to be gone from this base place'. He was detained here almost a month.

As court physician, Harvey accompanied the King for his Scottish coronation. It was characteristic of the man that his chief interest was not the ceremony, nor the theological disputes, but his own visit to the Bass Rock in order to find out some points with regard to the solan goose or gannet, which then, as now, congregated on that island in great numbers.

The clouds were gathering for the Civil War, and Harvey's official association with the King rendered him *non persona grata*, not only with the Parliamentarians in the House of Commons, who tried to oust him from St. Bartholomew's Hospital, but also to a mob who sacked and plundered his official lodgings in Whitehall. Harvey, referring to this outrage, says the cause of his sorrow is that certain rapacious hands 'stripped not only my house of its furniture but, what is subject to far greater regret with me, my enemies abstracted from my museum the fruits of many years of toil . . . . whence it has come to pass that many observations, particularly on the generation of insects, have perished with detriment, I venture to say, to the republic of letters'. It has been assumed that Harvey's medical observations, as well as some other papers, were destroyed by this mob.

Sitting under a hedge and reading a book, he was present at the battle of Edgehill—that strange encounter in which, the day before, an oblivious squire had gone hunting between the armies but had obligingly agreed, on having his position explained to him, to take a hand with his servants. Later, owing to a cannon ball and the fact that he was in charge of the future Charles II and his brother, Harvey, to his annoyance was obliged to move. But he seems to have played his part in succouring the wounded, of whom it is related that Adrian Scrope, having been left for dead, was afterwards 'recovered by the immortal Dr. Will Harvey'.

There must have been many of Harvey's friends on both sides of that great struggle, but there is no evidence that he took any part in political affairs whilst he was with the King at Oxford from 1642 to 1646. Here he was incorporated a Doctor of Medicine, and during the last year was appointed Warden of Merton College.

After the Civil War he retired, a saddened man of 68, to the quiet and rest provided him by his relatives in and about London; his rich and influential merchant brothers may well have secured him protection in his retirement.

It is probable that, from 1602 onwards, Harvey was working at his subject, and from 1609, when he was formally elected Physician to St. Bartholomew's, he must have gradually acquired a reputation as an anatomist as well as a physician. In 1615, at the age of 27, he was appointed Lumleian lecturer to the College of Physicians, a post which he held until 1656, the year before he died.

The 'Prelectiones' which bear the date of 1616 are really the lecture notes for the first anatomical course which Harvey delivered. The practical instructions which he laid down for his own guidance in method seem to be the last word on the proper objects and limits of oral teaching and demonstration, and are a model to be utilized by every medical teacher of our own time. The special value of this MS, which deals chiefly with the viscera, is that we have here Harvey's first account of the circulation of the blood, the full exposition of which he gave in his printed book in 1628.

I need not dwell on Harvey's masterpiece, so brief, so terse and so convincing, a model for all time of inductive reasoning. Let us never forget the two supreme doctrines which he established and which neither his predecessors nor his contemporaries had understood: Firstly, the circulation of the blood from the heart through the arteries and back through the veins to the heart again. The pulmonary circulation, it is true, had been more or less understood, but it was reserved to Harvey to establish the true nature of the systemic circulation, and to give the *coup de grace* to the ebb-and-flow movement of the blood current, which was the accepted explanation up to this time. And secondly, the muscular contraction of the ventricles, the real motive force of the circulation, which previously had been conspicuously ignored and misinterpreted.

Harvey's view of the respiratory function was inadequate, for he summed it up in cooling and ventilation, and never thoroughly faced the difference between arterial and venous blood. But the day of chemistry was not yet, and it was impossible for him, without the help of a microscope, to bridge over the space between the arteries and the veins.

His work on generation has been spoken of as Harvey's aftermath; it tells us incidentally about the worldly experiences of a long and chequered career, and displays Harvey's speculative and contemplative side.

From 1646 to 1657, his Court duties ended, his private practice relinquished, Harvey lived henceforward in retirement, first with one of his merchant brothers, then with another in London or in the country near by. His brothers were substantial and influential people and looked after his money affairs, so that he had no anxieties.

We know little of his married life, but it must have been a happy one. His wife was the daughter of an eminent physician, Dr. Lancelot Brown, and she predeceased him by some years. He was childless, but his care for his nephews and nieces was very marked. The College of Physicians was the chief interest of his old age. Besides being Lumleian lecturer, he had been at different periods censor, treasurer elect, and finally, when he graciously declined the presidency, Councillor of the College.

An interesting autographical note by William Heberden has been discovered on the habits of Harvey, communicated to him by Harvey's great-niece, as follows: 'In 1761 Mrs. Harvey (great niece to Dr. Harvey) told me that the Doctor lived at his brother's at Roehampton, the later part of his life. That he used to walk out in a morning combing his head in the fields. That he was humersome and would sit down exactly at the same time he had appointed for dinner, whether the company were come or not. That his salt cellar was always filled with sugar, which he used to eat instead of salt. That if the gout was very painful to him in the night, he would rise and put his feet into cold water.'

To read Harvey's work is to be conscious of companionship with a man of dynamic personality and a great lover of truth. Across three centuries of time William Harvey, the immortal founder of modern physiology and modern medicine, exhorts men to search and study out the secrets of Nature by way of experiment, and for the honour of the profession to continue in mutual love and affection. In the words of Hobbes, though he had lived to see 'his new doctrine everywhere established', he had been one of the few that had 'conquered envy'. And his last letter, perhaps, tells us why. It was to a young doctor who had sent him a specimen, and having thanked him for his kindness, since it was in just such deviations from the normal that Nature revealed her 'secret mysteries', he pointed out that as an explorer his own days were, alas, numbered. But 'it will always', he wrote, 'be a pleasant sight to see distinguished men like yourself engaged in this honourable arena' and, 'whatever you do', he added, 'not a very hard task, surely, still love yours most respectfully William Harvey'.

ASSOCIATION NEWS : VERENIGINGSNUUS

NATAL COASTAL BRANCH

REPORT OF HON. SECRETARY, DR. K. W. DYER

The following is an abstract of the Hon. Secretary's report for 1957:

After referring to the highly successful South African Medical Congress which was held in Durban in September 1957, the inspiring leadership of the President, Dr. H. Grant-Whyte, and the work so well carried out by the Chairman, Dr. A. Bloomberg, and his able band of colleagues, Dr. Dyer's report continued:

The following Office-Bearers and Members were elected to the Branch Council for the year 1957: *President* Dr. N. A. Rossiter, *Vice-President* Dr. N. R. Pooler, *Hon. Treasurer* Dr. F. Walt, *Hon. Secretary* Dr. K. W. Dyer, *Associate Secretary* Dr. D. Martyn; *Members of Council* Dr. S. Disler, Dr. R. Mundy and Dr. J. Stolp. Dr. L. Knox, as Immediate Past-President, remained an ex-officio member, still to brighten the dreary hour with his well-timed and 'lance-like' comments.

The following were Federal Council members for the first half of the year: Dr. A. Broomberg, Dr. E. W. S. Deale, Dr. H. Grant-Whyte, Dr. N. A. Rossiter and Dr. Alan B. Taylor. At the Federal Council elections in August, the following were returned: Dr. A. Broomberg, Dr. S. Disler, Dr. H. Grant-Whyte, Dr. N. A. Rossiter, Mr. A. G. Sweetapple and Dr. Alan B. Taylor. Dr. D. A. Edington was appointed to fill the vacancy created when Dr. Disler was elected as a Federal Councillor.

Dr. Rossiter is to be congratulated on a highly successful year of Chairmanship, in which he has handled many difficult matters with easy understanding.

Membership. The present membership of the Branch is 535.

Association Honours. We are proud to record two Association awards, confirmed by Federal Council, during the year:

Dr. A. Broomberg, Bronze Medal for long and meritorious service to the association. This was presented to him at the Opening Ceremony of Congress.

Dr. J. Drummond, Emeritus Membership for long and meritorius service to the Association. This Certificate was to be presented to Dr. Drummond at the Annual General Meeting on 13 February 1958. We regret sincerely to have to record his death on 25 January 1958.

Deaths. With regret we record the death also of Dr. G. S. Andrews, Dr. A. D. McKenzie and Dr. D. O'Donovan. To their relatives we extend our sincere condolences.

Branch Meetings

There were 3 business meetings of the Branch during the year: the first, an emergency meeting after the Annual General Meeting to discuss 'The Transfer of the Medical School'; at the second we were pleased to have Dr. Tonkin, the Medical Secretary,

with us to assist with his comments; the third was devoted mainly to the possibilities of establishing a Branch headquarters.

Branch Council

It is a pity that all members cannot see for themselves the volume of voluntary work which the Council does, not only in long night meetings, but in the various sub-committees.

The Ethical Committee has again happily had no case to consider.

The Contract Practice Sub-committee has as usual done an enormous amount of work in arbitrating between our members and medical aid and benefit societies, constantly striving to protect us and at the same time trying to ensure a fair deal for the societies. Our thanks go to Dr. E. W. S. Deale and Dr. A. Broomberg, Chairmen in succession, and to Dr. D. Martyn, Secretary, who has done a first-class job of continuous liaison work.

Sub-Committee for Integration of General Practitioners into Hospital Services. We have had no replies to the memoranda which were sent at the beginning of the year to the Director of Medical Services, Natal, and to the Joint Standing Advisory Committee of King Edward VIII Hospital, suggesting schemes to provide more opportunity for general practitioners to work at Addington and King Edward VIII Hospitals. We have recently again pressed for a reply.

Sub-committee for Deputation to Administrator re Invasion into Private Practice. A deputation from the Natal Inland Branch and our Branch was given a good hearing by the Administrator-in-Executive last February, but again frustratingly we have had no satisfactory answer. Our Sub-Committee, Dr. Grant-Whyte (President of the Medical Association of South Africa), Dr. Rossiter (President of the Natal Coastal Branch), Dr. Knox (Immediate Past President), Dr. Pooler (President-Elect) and the Natal Inland Branch Sub-committee are at present considering another memorandum to present to the new Administrator, based on the Provincial Auditors' recent Report of the uneconomical running of the Provincial hospitals. We believe this to be due to (1) failure to collect moneys due for hospitalization, and (2) the ineffective almoner system for both Europeans and non-Europeans, which allows free treatment for all classes of patients. A block to receive private patients, as is attached to all modern hospitals overseas, would mean a tremendous saving to the Province and also greatly benefit private practitioners. Accidents and emergency cases, who could afford private fees, and W.C.A. cases would be transferred to this block, which would also cater for specialist procedures of a highly technical nature which could not be undertaken in nursing homes.

Sub-committee for Establishment of Rehabilitation Centre. The Council are investigating the possibilities of instituting a pilot scheme for a Rehabilitation Centre at one of the present nursing homes, in which all interested specialists may take part, and general practitioners may attend their own cases. Such a centre, as exists overseas for accidents and rehabilitation, could be of great value to the public and the profession.

Branch Building. A sub-committee, convened by Dr. Walt, has done a tremendous amount of spade work in an attempt to establish a permanent home for our Branch headquarters. Where this will be, or what the method of raising the necessary funds, has not been finally decided.

Revision of Branch Rules. These have remained unaltered since 1932 and the preliminaries towards revision have already been completed by the Executive of Branch Council.

Sub-Groups

These have continued to function in the interests of their members and have kept the Council informed of their affairs. The Orthopaedic and Pathological Groups would appear to be the most active.

It is your Secretary's opinion that it is necessary that these Groups should continue to function, as they best can understand the difficulties of their respective members, but that any representations from Groups are most favourably received by Federal Council through the medium of Branch Councils, rather than *via* National Groups. It is thus important that a nice representative balance between the various specialties and general practitioners be maintained on the Council and that both general practitioners and specialists, particularly Federal Councillors, do all in their power to improve their understanding of the other's problem.

While it is realized that there is considerable extra cost, academic work and financial risk attached to specialization, which entitles the specialist to a higher rate of remuneration, it must also be known that today, with the enormous growth of the medical aid societies, with preferential tariff which limits the general practitioner's income yet increases the scope of the specialist, the honest GP in a large centre has no jam and only a modicum of butter on his bread. It is apparent that this acute economic factor has been responsible for most of the differences which have arisen since the War between these two interdependent branches of our profession.

Industrial Medicine Sub-Group. The Council is anxious that those Doctors who are interested in Industrial Practice should form themselves into a group to protect their interests and to formulate ethical rules for this type of practice. A group within the Association, 'The South African Society of Industrial Health' already exists and the Council will proceed with the formation of a local Sub-Group.

Personal

My sincere thanks to Mrs. Brinkworth for her much-needed encouragement and constructive criticism during the past 2 years of my Secretaryship. Her understanding and experience is invaluable to any Secretary.

To my successor my best wishes. I regard it as a privilege to have worked with the other Council members, and am grateful for the advice and assistance which has always graciously been given me.

In the past two years almost all Branch Council candidates have been unopposed, but it is gratifying to see that there will be a keenly contested election for 1958. I wish the successful candidates a still greater success in the advancement of our professional status in the eyes of the public, and in the improvement of conditions for full-time service and private practice. It is to this end that our Association exists.

REPORT OF HON. TREASURER, DR. F. WALT

Dr. Walt reported that for 1957 the audited accounts of the Branch showed a profit of over £200. The contributions to the Benevolent Fund totalled £102. The benefits paid out by the Fund to dependents of the Branch amounted to £510, and the objective of the Branch should be to raise £500 a year for the Benevolent Fund.

The Medical War Memorial Bursary Fund stood at £2,594. During its 10 years of existence only 3 beneficiaries had benefited, and Dr. Walt suggested that it could be put to better use if the objective of the Medical Building Subcommittee, which was formed last year to establish a permanent home for the Branch, was achieved. It was suggested that the building should be a war memorial and, tentatively, that its name might be 'The Medical Memorial Centre'.

The Durban Congress. The income (and expenditure) had almost reached £12,000 and, though final figures were not available, a profit of £250 was anticipated.

REPORT OF HON. SECRETARY OF CLINICAL SUBCOMMITTEE, DR. AUBREY BEILES

Dr. Beiles said that the Subcommittee was fortunate in having Dr. Pauline Klenerman as its Convener.

Apart from the South African Medical Congress, 7 clinical meetings had been held during the year. Of these, 3 were true clinical discussions at Addington Hospital, at King Edward VIII Hospital, and by the Department of Medicine of the Natal University, respectively. Except for the hospital meetings all the gatherings were held at the Medical School, and thanks were tendered to the Dean, Heads of Departments, the Registrar and the Caretaker for their kind help. Attention was called to the small attendance at clinical meetings.

Available to all Branch members were regular monthly meetings held by the (1) University Department of Medicine, (2) King George V Hospital, (3) Addington Hospital Departments of (*a*) Medicine and (*b*) Paediatrics, (4) Radiologists, King Edward VIII Hospital, and (5) University Department of Pathology.

AMENDMENTS TO WORKMEN'S COMPENSATION ACT TARIFF

Under section 79 of the Workmen's Compensation Act No. 30 of 1941, the Workmen's Compensation Commissioner gives notice in Government Notice No. 338 of 17 March 1958, that, after consultations with the Medical Association of South Africa, he has approved of the correction and amplification of the scale of fees published in Government Notice No. 1668 of 1956, with effect from 1 February 1958, as set out as follows:*

1. *Item* 8.

In sub-item (1) (*p*) insert the words 'including treatment of all associated fractures of the facial bones' after the word 'Maxilla'.

[*Item* 8 (1) (*p*) *refers to the treatment of simple fractures of the* '*Maxilla*'—Editor.]

In sub-item (1) (*r*) insert the word 'and/' after the word 'complete' and the words 'single or multiple fractures', in brackets, after the word 'fracture'.

[*This amendment applies the fees hitherto payable under* (*r*) *for* '*Mandible—complete or compound fracture*' *to* '*Mandible—complete and/or compound fracture* (*single or multiple fractures*)'—Editor.]

2. *Item* 12.

Delete the existing fees in sub-item (2) and substitute therefor 'see items 1, 4 (1) or 40 (1)'.

[*Item* 12 *refers to* '*treatment of joint injuries*', *and sub-item* (2) *thereof to* '*sprains of large joints*'. *Item* 1 *deals with* '*Visits and examinations* (*including ordinary treatment of single or multiple injuries*)'. *Item* 4 (1) *deals with* '*simple* (*single or multiple*) *wounds and bruises, burns and other slight injuries not otherwise specified, requiring not more than* 6 *attendances—without suture*'. *Item* 40 (1) *is as follows:* '*Fees for services not specified in this Scale of Fees, and fees for specially complicated or prolonged treatment shall be referred to the Commissioner for assessment*'.—Editor.]

In sub-item (6) insert the words 'inclusive of subsequent manipulations and/or aspirations of the joint' after the word 'joint'.

[*Sub-item* (6) *refers to* '*Open operation for internal derangement of knee joint*'.—Editor.]

In sub-item (11) (*b*) insert the words 'including epidural injections and/or local infiltrations' after the word 'jacket'.

[*Sub-item* (11) (*b*) *refers to* '*Conservative treatment with traction and/or P.O.P. jacket*'.—Editor.]

3. *Item* 13.

Delete the existing fees in sub-item (1) and substitute therefor 'see items 1, 4 (1) or 40 (1)'.

[*Item* 13 *deals with* '*Treatment of uninfected injuries of muscles and fascia*', *and sub-item* (1) *thereof with* '*treatment not involving open surgical operation* (*including injections*)'. *See also first note under* '2. *Item* 12' *above*.—Editor.]

4. *Item* 29.

The existing fees in sub-item (1) (*c*) to be raised to the level of that laid down in item 11 (2) (*q*), viz. £4 15s., £7, £7, and £10.

Item 29 *deals with* '*Physiotherapeutic treatments*', *and sub-item* (1) (*c*) *thereof with* '*Epidural injection*' (£2 12*s*. 6*d*., £4, £4, *and* £6). *Item* 11 (2) (*q*) *deals with* '*Epidural injection of Novocaine for sciatica*'.—Editor.]

In sub-item (2) insert the words 'or any other general practitioner or radiologist' after the word 'practitioner' where it appears in the third line of the relevant sub-item.

Sub-item (2) *is as follows* (*the word* '*practitioner*' *where it appears in the third line of the original text is here printed in Roman type:*

'(2) *Payment for physiotherapy administered by a non-specialist medical practitioner who is already in charge of the general treatment of the workman concerned or by any partner, assistant or employee of such* practitioner *shall be made only with the express approval of the Commissioner: Application for approval to be made in advance if possible. Wherever such approval is granted the fees payable in respect of the physiotherapy administered shall be reduced in accordance with the provisions of item* 39 (2)'. *Item* 39 (2) *is as follows:*

'(2) *Where secondary operations are performed by the same medical practitioner or his partner or assistant in medical practice, within twelve months after the primary operation,* 50 *per cent of the prescribed fee shall be payable in respect of such secondary operations.*'—Editor.]

5. *Item* 40.

Insert new sub-item—

(3) If the fee in the Scale of Fees is disproportionately high in relation to the actual service rendered, a special assessment may be made by the Commissioner.

[*The existing Item* 40 *is as follows:*

'(1) *Fees for services not specified in this Scale of Fees and fees for specially complicated or prolonged treatment shall be referred to the Commissioner for assessment.* (2) *Fees for the treatment of an injured workman whose death occurs prior to the completion of treatment for which there is a prescribed fee*(*s*) *shall also be referred to the Commissioner.*'—Editor.]

* A previous amendment in the scale was made in Government Notice No. 686 of 1957: see S. Afr. Med. J. (1957): **31**, 887.

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IN MEMORIAM

DR. LOUIS LAYTON

Dr. J. H. Marks, of Glen Harmony, O.F.S., writes: On 13 February 1958, Dr. Louis Layton died in Johannesburg.

Dr. Layton was born in Maritzburg in April 1885, and at the age of 13 was apprenticed to a chemist in Durban. He later went to Edinburgh and in 1914 graduated L.R.C.P., L.R.C.S. (Edin.), L.R.F.P.S. (Glasg.)

Dr. Louis Layton

On graduation he joined the R.A.M.C. and served in France during World War I. On demobilization he returned to South Africa and after working at the Johannesburg Hospital embarked on private practice in Johannesburg.

Dr. Layton was the real family doctor of the old school—the confidant and friend of his patients. His kindness and consideration brought pleasure to all who came in contact with him.

To those who knew and loved him his loss is great and his sterling qualities, his modesty and gentleness of spirit, remain as an endearing memory.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

VACANCY—ASSISTANT EDITOR

Applications are invited from medical practitioners for the post of Assistant Editor in the service of the Medical Association of South Africa at its Head Office in Cape Town.

The salary scale attaching to the post is £1,250×50—1,750 per annum, plus an annual cost-of-living allowance of £176 for single men and £352 for married men. The commencing salary will be determined according to experience.

The successful applicant must contribute to the Association's Superannuation Fund. He will also be expected to assume duty as soon as possible after appointment.

Applications must reach the Secretary, Medical Association of South Africa, P.O. Box 643, Cape Town, before 31 March 1958.

A. H. Tonkin
Secretary

Medical House
Cape Town
24 February 1958

VAKATURE—ASSISTENT-REDAKTEUR

Aansoeke word van geneeshere ingewag vir die betrekking van Assistent-Redakteur in diens van die Mediese Vereniging van Suid-Afrika, by die Hoofkantoor te Kaapstad.

Die salarisskaal aan die pos verbonde is £1,250×50—1,750 per jaar, plus 'n jaarlikse duurtetoeslag van £176 vir 'n ongetroude en £352 vir 'n getroude man. Die aanvangssalaris sal volgens ondervinding bepaal word.

Die suksesvolle kandidaat moet by die Vereniging se pensioenskema aansluit. Hy sal ook verwag word om so spoedig moontlik na aanstelling diens te aanvaar.

Aansoeke moet die Sekretaris, Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, bereik vóór 31 Maart 1958.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
24 Februarie 1958

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

ERGODRYL

Parke, Davis Laboratories (Pty.), Ltd., have introduced Ergodryl a new oral preparation for the treatment of migraine. They supply the following information:

Ergodryl is a non-narcotic agent for the treatment of migraine and vascular headaches, each capsule containing 1 mg. of ergotamine tartrate, 100 mg. of caffeine citrate, and 25 mg. of diphenhydramine hydrochloride (Benadryl).

Ergotamine tartrate exerts a vasotonic action, especially marked on the extracranial arteries. By its vasotonic effect it reduces the amplitude of the pulsations in these vessels and so the intensity of the headache is decreased.

Caffeine citrate stimulates the central nervous system and potentiates the action of ergotamine. Therapeutic doses of caffeine have two vascular effects, viz. (*a*) peripheral vasodilation (direct action on vascular wall), and (*b*) central vascoconstriction due to stimulation of the vasomotor centres.

Benadryl, because of its antispasmodic and antihistamine effect, has proved valuable in many cases of migrainous attacks, which are considered due to a mechanism similar to histamine-induced vasodilation of the branches of the external carotid.

Indications. The principal indications are genuine migraine, migraine in children and histaminic cephalalgia.

Dosage and Administration. Ergodryl should be taken as early as possible in the prodromal phase. With the first attack treated with Ergodryl one capsule should be given as early as possible. If the headache is not aborted, one further capsule may be given every 30 minutes up to a total of 6 capsules. Once the dosage necessary to abort the attack has been determined, the patient can take the total number of capsules as a single dose on the first warning of an attack. However, no more than 4 capsules should be taken as a single dose. The patient should be advised not to take more than 10 capsules in any one week. Optimal results depend on dosage adjustment. The great advantage of oral Ergodryl therapy are that the patient can take the dose as he feels an attack is imminent and that the dosage can be adjusted to the intensity of the attack.

Contra-indications. Peripheral vascular disease, angina pectoris, impaired hepatic or renal function and pregnancy.

Ergodryl capsules are available in vials containing 10 capsules and in bottles of 100 capsules.

PASSING EVENTS : IN DIE VERBYGAAN

Radiological Society of South Africa. The Transvaal Sub-group of this Society, at a General Meeting on 24 February 1958, elected the following office-bearers for the year 1958: *Chairman*, Dr. M. Fainsinger; *Vice-chairman*, Dr. C. Komins; *Secretary*, Dr. J. A. Louw; *Treasurer*, Dr. J. Friedman; *Members*, Dr. P. J. Loots, Dr. I. A. Brotman and Dr. J. St C. Ballenden.

* * *

Urological Congress, Cape Town, July 1958. The second independent meeting of the Urological Association of South Africa will be held in Cape Town from Wednesday to Saturday 16-19 July 1958. The main theme for discussion will be 'Tumours of the Bladder'. All interested practitioners are welcome to attend and information can be had from the Honorary Secretary, Dr. P. J. M. Retief, 514 Netherlands Bank Buildings, Cape Town.

* * *

The Swiss Federal Institute of Technology, Zürich, is prepared to offer a scholarship to a student holding a bachelor's degree at a South African University for advanced study at the Institute. The scholarship will be obtained in monthly instalments of 330-350 Swiss francs (about £27-£29) and includes exoneration from class fees. Bursary holders will be required to pay a compulsory tax of about 30 Swiss francs (£2 10s. 0d.) per semester. The scholarship will be valid for the duration of one or two semesters. Travelling expenses between South Africa and Switzerland must be borne by bursary holders themselves. The academic year begins in the middle of October and ends in the second half of July. It includes two semesters (1) from October to February and (2) from April to July. The purpose of the scholarship is to enable South African students to extend, during one or two semesters, their professional knowledge at the Federal Institute of Technology; it is not for the purpose of obtaining another degree. Studies can in principle begin in October as well as in April, or under special circumstances even during a semester (for instance on 1 January). Amongst the schools of the Institute are the following: Chemistry, Pharmacy, Mathematics and Physics, Natural Sciences, and a General Section of courses on history, philology, philosophy, politics and economics. The names of the schools do not include Medicine. Housing facilities are available at the Student Hostel of the Institute, boarding houses and private families, the expenses of board and lodging varying from frs. 250 (about £20) to frs. 300 (about £24 per month). Application forms and particulars are obtainable from the Division of Cultural and External Relations, Department of Education, Arts and Science, Private Bag 122, Pretoria; and completed applications on the prescribed forms must reach the Division not later than 19 April 1958.

* * *

Union of South Africa. Department of Health. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 7 March to 13 March 1958.

Plague, Smallpox, Typhus Fever. Nil.

Poliomyelitis

	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal	–	2	–	–	2
Cape Province ..	1	–	1	–	2
Orange Free State	1	1	–	–	2
Natal	2	–	–	1	3
Totals	4	3	1	1	9

Local Authorities		*Eur.*	*Non-Eur.*
Transvaal:			
Brakpan Municipality	U		1
Brits Municipality	U		1
Cape Province:			
Alicedale Village Management Board	U	1	
Worcester Municipality ..	U		1
Orange Free State:			
Hertzogville Municipality ..	U	1	
Kroonstad District	R		1
Natal:			
Durban Borough	U		1
Hilton Road Health Committee	U	1	
Kloof Health Committee ..	U	1	

U = Urban. R = Rural.

BOOKS RECEIVED : BOEKE ONTVANG

Biochemical Investigations in Diagnosis and Treatment. 2nd Edition. By John D. N. Nabarro, M.D., F.R.C.P. Pp. xi + 299. 25s. net. London: H. K. Lewis & Co. Ltd. 1958.

Handbook of Histopathological Technique. (including Museum Technique). By C. F. A. Culling, F.I.M.L.T., F.R.M.S. Pp. x + 446 + (27). 79 Figures. 51s. 9d. + 1s. 8d. postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

Burns—Pathology and Therapeutic Applications. By Simon Sevitt, M.D., M.Sc., M.A., F.R.C.P.I., D.P.H. Pp. xii + 364. 85 Figures. 43s. 3d. + 1s. 7d. Postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

The British Encyclopaedia of Medical Practice. Including Medicine, Surgery, Obstetrics, Gynaecology and other Special Subjects. *Medical Progress* 1957. Editor in Chief, The Lord Cohen of Birkenhead, M.D., D.Sc., LL.D., F.R.C.P., F.A.C.P., F.F.R. Pp. vi + 339 + (14). London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

Bone Diseases in Medical Practice. By I. Snapper, M.D., Pp. x + 229. 48 Plates. $15.00. New York and London: Grune & Stratton, Inc. 1957.

An Introduction to Medical Laboratory Technology. 2nd Edition. By F. J. Baker, F.I.M.L.T., F.I.S.T., F.R.M.S., R. E. Silverton, A.I.M.L.T., F.R.M.S. and Eveline D. Luckcock, A.I.M.L.T. Pp. xiv + 408. 118 Figures. 43s. 3d. +

1s. 7d. Postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

Chronic Bronchitis, Emphysema, and Cor Pulmonale. By C. H. Stuart-Harris, M.D. (London), F.R.C.P. and T. Hanley, M.D. (Lond.), M.R.C.P. Pp. vi + 246. 61 Figures. 42s. 0d. + 1s. 3d. Postage. Bristol: John Wright & Sons Ltd. 1957.

Recent Trends in Chronic Bronchitis. Edited by Neville C. Oswald, M.D. (Cantab.), F.R.C.P. (Lond.), Pp. vii + 199. 74 Figures. 30s. net. London: Lloyd-Luke (Medical Books) Ltd. 1958.

Non-Venereal Syphilis—A Sociological and Medical Study of Bejel. By Ellis Herndon Hudson, M.D., D.T.M. & H. (Lond.), F.A.C.P. Pp. viii + 204. 81 Figures. 30s. net + 1s. 2d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

Cerebral Palsy in Childhood. By Grace E. Woods, M.D., D.P.H., D.C.H. With a Foreword by Peter Henderson, M.D., D.P.H. Pp. xii + 158. 41 Figures. 27s. 6d. + 1s. 0d. postage. Bristol: John Wright & Sons Ltd. 1957.

The Physiological Basis of Gastro-intestinal Therapy. Selected Topics. By Heinrich Necheles, M.D., Ph.D., F.A.C.P. and Martin M. Kirshen, M.D., F.A.C.P. Pp. v + 330. $8.75. New York and London: Grune & Stratton, Inc. 1957.

Management of the Handicapped Child. Diagnosis, Treatment and Rehabilitation. H. Michal-Smith, Ph.D., Editor. Foreword by Arnold Gesell, M.D., Pp. x + 276. Illustrations. $6.50. New York and London: Grune & Stratton, Inc. 1957.

Year Book of General Surgery—1957—1958. Edited by Michael E. de Bakey, B.S., M.D., M.S. With a Section on *Anesthesia*, edited by Stuart C. Cullen, M.D. Pp. 560. 128 Figures. $7.50. Chicago: Year Book Publishers, Inc. 1957.

Paediatrics for Nurses. 2nd Edition. By Arthur G. Watkins, M.D., F.R.C.P. Pp. 200. 22 Figures. 15s. 0d. + 10d. postage. Bristol: John Wright and Sons Ltd. 1958.

Allergy in Pediatric Practice. By William B. Sherman, M.D. and Walter R. Kessler, M.D., Ph.D. Pp. 296. Illustrated. South African Price: £3 18s. 9d. St. Louis: The C. V. Mosby Company. 1957.

Psychology and Psychological Medicine for Nurses. By Portia Holman, M.A., M.D., M.R.C.P., D.P.M. Pp. 156. 10s. 6d. net. London: William Heinemann—Medical Books—Ltd. 1957.

Cortisone Therapy. Mainly Applied to the Rheumatic Diseases. By J. H. Glyn, M.A. (Cantab.), M.D., M.R.C.P., D. Phys. Med. Pp. x + 162. 4 Figures. 21s. net. London: William Heinemann—Medical Books—Ltd. 1957.

A Contribution to the Study of Portal Hypertension. Jacksonian Prize Essay Royal College of Surgeons 1956. By Alan Henderson Hunt, M.A., D.M., M.Ch., F.R.C.S. Pp. xi + 230. 120 Figures. 40s. net + 1s. 9d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

General Pathology. 2nd Edition. Edited by Sir Howard Florey, M.D., F.R.C.P., F.R.S. Pp. xv + 932 . Illustrated. 84s. net. London: Lloyd-Luke (Medical Books) Ltd. 1958.

Stedman's Medical Dictionary. 19th Revised Edition. Edited by N. B. Taylor, V.D., M.D., F.R.C.S., F.R.C.S. (Edin.), F.R.C.P. (Can.), M.R.C.S. (Lon.). Pp. xlvi + 1656. 88s. London: Ballière, Tindall & Cox, Ltd. 1957.

Gastro-intestinal Obstruction. By Meyer O. Cantor, M.D., M.S., F.A.C.S. and Roland P. Reynolds, M.D., F.A.C.S. and 3 other contributors. Pp. xii + 566. 415 Illustrations and Drawings. 144s. Baillière, Tindall and Cox Ltd. 1957.

Chromotographic Techniques. Clinical and Biochemical Applications. By Iver Smith, B.Sc., Ph.D., F.R.I.C. and 9 other authors. Pp. xiii + 309. Illustrated. 45s. net. London: William Heinemann. 1958.

The Door of Serenity. By Ainslie Meares, M.B.B.S., B.Agric.Sc., D.P.M. Pp. 110. Coloured Plates 24. 21s. net. London: Faber & Faber. 1958.

High Arterial Pressure. By F. H. Smirk, M.D., F.R.C.P., F.R.A.C.P. Pp. xxxvi + 764. Illustrated by figures and plates. 75s. Oxford: Blackwell Scientific Publications. 1957.

Polymyositis. By John N. Walton, M.D., M.R.C.P. and Raymond D. Adams, M.D. Pp. x + 270. Plates 47. 32s. 6d. net. Postage abroad 1s. 3d. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

British Pharmacopoeia 1958. Published under the direction of the General Medical Council. Pp. xxvi + 1012. 63s. net. London: The Pharmaceutical Press. 1958.

Medical Teleology and Miscellaneous Subjects. By F. Parkes Weber, M.A., M.D., F.R.C.P., F.S.A. Pp. viii + 86. 15s. net. London: H. K. Lewis & Co. Ltd. 1958.

Die Physiologie des Lymphozytenwechsels und seine Beeinfluhbarkheit durch Hormone des Hypophysen-Adrenal-systems. Von Priv. Doz. Dr. H. G. Hansen. 1958. viii, 164 Seiten. 74 teils mehrfarbige Abbildungen Gr.-8, kartoniert M 18.50. Stuttgart: Georg Thieme Verlag. 1958.

Bergey's Manual of Determinative Bacteriology. By Robert S. Breed, E. G. D. Murray, Nathan R. Smith assisted by Ninety-four contributors. Seventh Edition. Pp. xviii + 1094. 120s. London: Baillière, Tindall and Cox Ltd. 1957.

Frazer's Anatomy of the Human Skeleton. Fifth Edition. Edited by A. S. Breathnach, M.D., M.Sc. Pp. viii + 247. Illustrations 197. Many in Colour. 50s. net. London: J. & A. Churchill Ltd. 1958.

REVIEWS OF BOOKS : BOEKRESENSIES

CALCIUM METABOLISM

Calcium Metabolism. By J. T. Irving, M.A., Ph.D., M.D., F.Odont.Soc.S.A. Pp. xi + 177. 11s. 6d. net. London: Methuen & Co. Ltd. New York: John Wiley & Sons. Inc. 1957.

Contents: Preface. I. Introduction. II. Content and Availability of Calcium in Foods. III. Intestinal Absorption of Calcium. IV. Intestinal Absorption of Calcium (continued). Influence of Phytic Acid. V. Intestinal Absorption of Calcium (continued). The Action of Vitamin D. VI. Calcium Content of the Body and of Various Tissues. VII. Utilization and Retention of Calcium in the Body. VIII. The Dietary Requirement of Calcium. IX. The Calcium of the Blood. X. Bone Formation and the Influence of Various Factors upon this. XI. Bone—Chemistry and Physics. XII. Enzymes and Bone Formation. XIII. The Calcium Metabolism of the Teeth. XIV. The Excretion of Calcium. XV. Extra-skeletal Functions of Calcium. Summary. Index.

The subject matter of this book is based on a course of advanced lectures given by the author when Professor of Physiology at the University of Cape Town. A general review of calcium metabolism was long overdue and this monograph is a welcome addition to an excellent series on biochemical subjects by an acknowledged auhority. The author points out in the preface that the clinical aspects of calcium metabolism have not been dealt with except when these illustrate physiological principles as in rickets and diseases of the parathyroid gland. The clinician will, nevertheless, find much of great value in a book primarily written for physiologists and biochemists.

Some new and important conclusions are arrived at. In man the only important single factor to affect calcium absorption in the intestine is vitamin D. It is doubtful if phytic acid, which experimentally depresses calcium absorption, plays such a role in practical dietetics. The author presents a very good case for the view that the previous calcium intake is, within wide limits, the most important controlling factor in determining the level of calcium intake for equilibrium, owing to the ability of the body to adapt to low dietary levels of calcium. It follows, therefore, that the calcium requirement is a function of previous dietary history. With regard to the action of the parathyroid gland in controlling the calcium level of the blood the balance of evidence supports the view that the gland acts on calcium mobilization and not on renal phosphate excretion. It now seems certain that Robison's alkaline phosphatase is concerned solely with matrix formation and not with calcification.

The subject matter is clearly presented and a full list of references is given at the end of each chapter. The author is to be congratulated on his comprehensive and essentially up-to-date review of the subject. The reviewer warmly recommends it to the attention of all who wish to acquire information on modern aspects of calcium metabolism.

J.H.

SURGICAL PRINCIPLES

Surgery Principles and Practice. Edited by J. Garrott Allen, M.D., Henry N. Harkins, M.D., Ph.D., Carl A. Moyer, M.D., Jonathan E. Rhoads, M.D., D.Sc. With 26 contributors. Pp. xxii + 1495. Illustrations 621. £5 10s. net. London: Pitman Medical Publishing Co., Ltd. 1957.

Contents: 1. Surgical Philosophy. 2. Wound Healing. 3. Applied surgical Bacteriology. 4. Surgical Infections. 5. Fluid and Electrolytes. 6. Nutrition. 7. Shock. 8. Blood Transfusions and Allied Problems. 9. The Principles of Isotope Technics in Surgery. 10. Neoplastic Disease—General Considerations. 11. The Assessment of Operative Risks. 12. Anesthesia. 13. Operative Surgical Care. 14. Non-operative Surgical Care. 15. The Endocrine and Metabolic Basis of Surgical Care. 16. Burns. 17. Radiation Injury from Local or Total Body Exposure. 18. Fractures and Dislocations: General Considerations. 19. Fractures and Dislocations of the Upper Extremity. 20. Fractures and Dislocations of the Lower Extremity. 21. Fractures and Dislocations of the Spine, the Pelvis, the Sternum and the Ribs. 22. Principles of Hand Surgery. 23. Military Surgery. 24. Skin and Subcutaneous Tissues. 25. Breast. 26. Thyroid, Thymus and Parathyroids. 27. Pituitary and Adrenal Glands. 28. Esophagus. 29. Stomach and Duodenum. 30. Liver, Gallbladder and Bile Passages. 31. Pancreas. 32. Spleen. 33. Mesentery, Splanchnic Circulation, Portal Hypertension and Mesenteric Thrombosis. 34. Appendicitis and the Acute Abdomen. 35. Peritoneum, Peritonitis and Intra-Abdominal Abscesses. 36. Anatomy, Physiology and Treatment of Intestinal Obstruction. 37. The Intestinal Tract (continued). 38. Anorectum. 39. Hernia. 40. Peripheral Vascular Surgery. 41. Cardiac Surgery. 42. Lung. 43. Pulmonary Tuberculosis. 44. Carcinoma of the Lung and Tumour of the Thorax. 45. Principles of General Plastic Surgery. 46. Pediatric Surgery. 47. Gynecology. 48. Urology. 49. Orthopedics (Nontraumatic). 50. Surgery of the Nervous System. Index.

In a surgical world where most books for undergraduates tend to be rather conventional and therefore redundant, it is refreshing to encounter a new text book of surgery designed by four men who are lucid advocates of the modern approach to surgery.

As active Professors in leading American medical schools, they recognise the existence of excellent text-books in which may be found details of invidivual disease processes. While these printed products of what a recent visitor referred to as the 'pre-milli-equivalent age' remain invaluable, their experience as teachers has induced them to compile a book especially designed for the 'post milliequivalent student'. In doing so, they have chosen to plan the text mainly as a scaffolding on which the student may build using the material provided by the excellent bibliography.

The opening chapter, entitled 'Surgical Philosophy', reflects the stimulating spirit of the book. The fundamentals of surgery such as the problems of Shock, Infection and Inflammation are emphasized, but the unique quality of the text lies in the basic discussions of modern subjects such as isotopes, fluid balance, radiation hazards, military surgery, nutrition and the metabolic response to surgery. The more conventional chapters on General Surgery, Orthopaedics and Urology do little more than outline current American thought on these topics without providing detail. One hesitates to criticise this as an omission as the authors probably regard this as outside the scope of this work. In a pioneering venture of this kind, it is difficult to strike a balance and impossible to satisfy all.

This book, compiled primarily to stimulate the intelligent student to read further and to appreciate the vital role of the basic sciences in surgery today, deserves to be read widely. It has a distinctive informality, however, and will not appeal to those who aim to use it as a detailed reference book or for the purposes of passing any specific examination.

W.A.J.

OFFICE CYTOLOGICAL TECHNIQUES

Cytologic Technics for Office and Clinic. By H. E. Nieburgs, M.D. Pp. viii + 233. 171 Figures. 87·75. New York and London: Grune & Stratton, Inc. 1956.

Contents: Preface. Introduction. 1. Oral Cavity. 2. Laryngeal Smears. 3. Cytologic Diagnosis of Cancer of the Maxillary Sinuses. 4. Cytologic Diagnosis of Pulmonary Carcinoma. 5. Esophagus. 6. Stomach. 7. Duodenal Drainage. 8. Descending Colon and Rectum. 9. Vulva. 10. Cervix Uteri. 11. Prognosis of Cervical Tissue Response to Radiation. 12. Endometrium. 13. Carcinoma of the Fallopian Tube. 14. Ovarian Carcinoma. 15. Office Procedures for the Estimation of Hormonal Activity. 16. Breast. 17. Serous Effusions. 18. Urine. 19. Prostate. 20. Skin. 21. Histocytology and Operating Room Procedures. 22. Mailing of Specimens. 23. Organization and Function of Cytologic Laboratory. 24. Inter- and Intradepartmental Co-ordination. 25. Considerations on the Training of Cytotechnicians, Cytologists and Cytopathologists. 26. Laboratory Procedures. References. Appendix. Index.

This is an essentially practical book dealing primarily with the methods of collecting material from those body cavities and organs to which the technique of cytological examination can be applied. The list of contents indicates how wide a field is covered and detailed instructions are given as to how specimens should be collected from these various sites.

The methods of preparing the specimens for examination and the staining techniques in use today are described in detail and there are chapters on the organization of a cytological laboratory, the mailing of specimens, and the training of cytological technicians.

Purely cytological aspects and the problems of cytological interpretation are not discussed at any length but the author does illustrate and mention in brief some of the cellular abnormalities encountered in neoplastic and other pathological processes in the different parts of the body.

This should certainly prove a useful book to the clinician who has to collect specimens for cytological diagnosis and to the technician who has to prepare these specimens for cytological examination. It makes no pretence to be a textbook of cytodiagnosis.

M.S.

HEALTH INSPECTOR'S HANDBOOK

The Public Health Inspector's Handbook. A Manual for Public Health Officers. Ninth Edition. (Completely Revised). By Henry H. Clay, F.R.S.H., F.I.P.H.E., assisted by Ronald Williams, O.B.E., D.P.A., F.R.S.H. Pp. xx + 614. 101 Illustrations. £2 0s. 0d. net. London: H. K. Lewis & Co. Ltd. 1957.

Contents: I. The Evolution of Public Health Law. II. Appointment, Designation, Duties, Training, and Qualification of Public Health Inspectors. III. Powers of Entry. IV. Nuisances. V. Smoke Abatement. VI. Offensive Trades. VII. Inspection of Premises. VIII. Housing—Procedure under the Housing Acts. IX. Housing—Premises Subject to Special Provisions. X. The Hygiene of Building. XI. Natural Lighting. XII. Water. XIII. Drainage. XIV. Sanitary Fittings and Apartments. XV. Plans. XVI. Conservancy and Sewage Disposal Camp Sanitation. XVII. Refuse Disposal and Public Cleansing. XVIII. Ventilation. XIX. Heating, Hot-water Supply, and Gas Services. XX. Factories, Workplaces, and Shops also Rag Flock, etc., Premises. XXI. Milk and Dairies. XXII. Food Control; Composition, Description, and Protection of Food. XXIII. Preservation of Food. XXIV. Slaughter-Houses and Knackers' Yards. XXV. Meat Inspection. XXVI. Fish Inspection. XXVII. Control of Infectious Diseases. XXVIII. Disinfestation. XXIX. Canal Boats. XXX. Elementary Vital Statistics. XXXI. Office Organisation and Routine. XXXII. Useful Memoranda and Glossary of Building Terms. Index.

This publication, which has now reached its ninth edition, has, as the title indicates, been written primarily for the Health Officials of the United Kingdom, it incorporates the latest health legislation promulgated since 1954 when the eighth edition was printed.

The book is readable, well printed, contains many excellent illustrations and covers, in an admirable manner, a very wide field.

I am doubtful whether it will find a place on the bookshelves of local health officials on account of the difference between our health legislation and that of the United Kingdom. For a reference work, however, it should be found more than useful for those persons responsible for environmental sanitation and its allied problems.

E.D.C.

JAMA CLINICAL ABSTRACTS

J.A.M.A. Clinical Abstracts of Diagnosis and Treatment. Pp. vi + 564. 85·50. New York and London: Intercontinental Medical Book Corporation with Grune & Stratton, Inc. 1957.

Contents: Preface. Digestive System. Respiratory System. Cardiovascular System. Urinary System. Reproductive System: Pregnancy, Breast, Ovaries, Vagina and Uterus, Testes. Endocrine System. Bones, Muscles, Joints, Connective Tissue. Blood and Lymphatic System. Skin. Nervous System. Eye, Ear, Nose and Throat. Poisonings. Infections. Miscellaneous Diagnosis and Treatment: Diagnostic Technics, Radiation, Drugs and Drug Reactions, Hypothermia. Index of Subjects.

This book is a collection of abstracts covering the main branches of clinical medicine with the emphasis on diagnosis and treatment. These abstracts were originally published in the Journal of the American Medical Association in the "Medical Literature Abstracts" section. This collection has been made from more than 1,200 articles and they are here arranged systematically on an organ or disease basis to allow of easy reference. A year's selected reading is thus made available, crystallizing the recent advances or fashions in clinical medicine.

There is no doubt that this book serves a valuable purpose in facilitating the task of the specialist and general practitioner in keeping abreast of clinical medicine amidst the ever-increasing flow of publications. It is another digest. Is it a justifiable addition to those already available in the English language?

It is an excellent selection showing the wisdom of the author and his colleagues, who, with their judgement and skill in choice and summarising, have produced a balance between the ultra-conservative and the over-enthusiastic appraisal of new medical articles. Digests of clinical medicine inevitably contain abstracts of many papers in which the conclusions are based on poorly controlled studies. Much that is claimed will soon be disproved or disclaimed. Perusal of these condensations indicates many such investigations. This however, does not detract from the value of these annuals. Clinical medicine must of necessity advance in a somewhat unscientific way, as strict control of the trials and experimental situations is often impossible. The conclusions provide a stimulus and if the work is performed in institutions of repute and the writings selected by experienced teams, the reader may peruse them with confidence.

One can recommend this book to the advanced medical student, the post-graduate student, the graduate nurse, the general practitioner and the specialist. It should be a valuable annual addition to their bookshelves.

J.H.G.

CORRESPONDENCE : BRIEWERUBRIEK

THE AGE OF THE FOETUS

To the Editor: I was surprised at the interpretation placed upon the closing paragraph of my letter[1] to you by Dr. McLachlan.[2]

I wrote in criticism of the quality of appraisal in your Editorial because I felt that this might encourage widespread recourse to radiological estimates of maturity, and in conclusion I outlined briefly the *possible* effects thereof upon (*a*) the mother, (*b*) the accoucheur and (*c*) the radiologist. The effect upon the last I thought might be 'additional work (and possibly additional remuneration) at the expense of frequent self-delusion'.

Dr. McLachlan's interpretation of the above is that this remark implies 'that radiologists are in the habit of examining patients purely for gain, without regard to the clinical indications'. This interpretation is a complete *non sequitur* and—I assure him—entirely contrary to my meaning. Furthermore, in order to avoid any continued misunderstanding, I apologise and wish to alter my phraseology to read as follows: 'offers to the mothers additional and usually unnecessary—expense'.

I trust that Dr. McLachlan will appreciate my desire to retain the end of the sentence, however, to the effect that 'radiologists (in providing a foetal-maturity estimation service) would do so at the expense of frequent self-delusion', for I feel that not only do many radiologists continue offering widespread advice (to patients as well as doctors) as to *how* delivery should be effected (with their 'forecasts' of the outcome of labour) but they now propose to inform accoucheurs *when* to do it as well!

Dr. E. H. Burrows[3] lists a number of arguments against my letter which stem mainly from lack of knowledge of this specialized subject combined with misquotation of sections of my letter. May I reply to these seriatim:

(1) In attempting to discount my contention that Dr. Blair Hartley's results[4] are largely a personal success and the result of an extremely high standard of radiography, Dr. Burrows maintains that 'Blair Hartley's methods could be practised (and his results equalled) anywhere in the world'. With a further few strokes of his pen, he attempts to ridicule my statement that the standard of radiography required is only attainable in a specialized X-ray department (such as Blair Hartley's) with modern high-output X-ray machines, first-class intensification screens and a limited volume of work.

No one would dispute that a specialized unit in South Africa *could* embark upon the same research work as Dr. Blair Hartley and probably achieve similar results, but what concerns the accoucheurs and mothers of South Africa is the efficacy of the service provided when Dr. Blair Hartley's methods are projected into universal usage. These, I assure your readers, Sir, would be inferior.

With regard to the standard of radiography, I have noted that in the following passage the Editor of the *Lancet*[5] gives me strong support in contradiction of Dr. Burrows' views: 'A very high standard of radiographic technique is required and this was the foundation of his success; for developing foetal epiphyses are not easily seen and they need a radiograph of the highest quality on every occasion.'

(2) The second point in my letter to the effect that 'special compression of the maternal abdomen is usually necessary before pilot A-P and P-A views are taken and an additional appropriate oblique view is then usually necessary' (for 'precise estimation of foetal maturity from radiographs taken for other purposes, e.g. cephalo-pelvimetry, will often prove unreliable') is distorted by Dr. Burrows as follows: 'It does not follow that because the technique is a specific one, it cannot be carried out by other workers elsewhere.' There was never any mention made of possible inability of others to use this method, but my point remains that it is a specialized method requiring additional radiographers and radiologists' time, additional X-rays and additional expense.

Again Dr. Burrows attempts to discredit my criticism of the fact that radiological estimation of foetal maturity is particularly unreliable in patients suffering from hydramnios or toxaemia by stating that Dr. Blair Hartley emphasizes this point. Surely acknowledgement of defeat is not synonymous with victory, Surely in post maturity, together with these two abnormalities. we have before us the array of circumstances under which a clinician is likely to ask for radiological estimation of maturity—albeit under exceptional circumstances at that!

Concerning the radiological diagnosis of postmaturity, I remarked that this 'requires special skill and is beset with pitfalls, being impressionistic and based upon consideration of a number of factors', Dr. Burrows omits the word postmaturity in an attempt to weaken my criticism. Surely he does not believe that the radiological diagnosis of postmaturity is simply made by identifying a developed ossification centre and clinical diagnosis by laying hands on the fundus uteri in postmaturity!

Dr. Burrows reaches his climax by describing me as grasping at radiation hazards as a final big stick with which to beat the radiologists. In actual fact my terminology was, 'Radiological diagnosis of postmaturity offers to mother and babe the *possibility* of radiation hazard and unnecessary obstetric interference'.

In conclusion he quotes Witts[6] as stating, 'We must balance the risks of X-ray diagnosis and therapy against their benefits' without having the remotest idea of what 'their benefits' are in the problem under discussion. To believe—as Dr. Burrows does—that the maternal mortality could be reduced by radiation estimation of maturity reflects the extent of his knowledge on this subject. His further belief that the mortality of the child may be 'significantly' reduced by such means is not far short of equidistant from the truth.

Derk Crichton
Professor of Gynaecology
University of Natal
Durban
17 March 1958

1. Crichton, D. (1958): S. Afr. Med. J., 32, 144.
2. McLachlan, F. W. (1958): *Ibid.*, 32, 259.
3. Burrows, E. H. (1958): *Ibid.*, 32, 259.
4. Hartley, J. B. (1957): Brit. J. Radiol., 30, 561.
5. Editorial (1958): Lancet, 1, 249.
6. Witts, L. J. (1957): Brit. Med. J., 1, 1197.

HYPNOTHERAPY IN CHILDREN

To the Editor: In the last few years hypnotism has become 'respectable', and its application to a number of medical problems is being increasingly studied. While its use in fields such as obstetrics and dentistry is well known, and much discussed in the literature, comparatively little attention has been devoted to hypnotherapy in children. This is surprising, for not only can hypnotherapy be successfully carried out in most children over

6 years of age, but it can be used with good effect in the treatment of behaviour problems and other disorders commonly found in children.

A useful introduction to the subject is provided by Dr. Gordon Ambrose's 'Hypnotherapy with Children' (Staples Press, London). Dr. Ambrose, who has had a wide experience in Child Guidance and psychiatric work, outlines the theory and history of hypnotherapy, the techniques employed, and their applications to certain paediatric problems.

Of the many children taken to the family practitioner for emotional difficulties, some have superficial emotional problems, and others require psychiatric treatment. If the children who fall between these two groups cannot receive treatment, says Dr. Ambrose, they will get worse, and they represent a serious challenge to modern medicine. The aim of the book is 'to help students and doctors to recognize the middle group, and to ensure rapid and effective treatment'.

In the section on treatment, particular attention is paid to a number of paediatric disorders characterized by *tension*. These are: Anxiety and delinquency; bed-wetting, asthma, epilepsy and stammering; and nail-biting, tics and habits. They are described under the headings of tension-conduct, tension-spasm, and tension-action, respectively. Hypnotherapy, by relieving the tension, helps the child to adjust adequately and quickly.

As the child's tension and anxiety are so often the result or a projection of such a state in the parents, treatment of the child is integrated with a particular approach to the parents. It is found that parental anxiety usually diminishes rapidly as the child improves, but where it remains serious hypnotherapy of the parents (usually the mother) is recommended by Ambrose. Case-histories are quoted to show the author's approach, and they are interesting and illuminating.

In working on these problems for the last few years, one has been able to confirm that hypnotherapy can be successfully carried out in most children over 6 years of age, and that it is useful in the treatment of tension-states in children, particularly for the 'middle group' referred to by Ambrose. In one's own experience, rapid improvement can usually be expected in tics and habits, anxiety states and fears. In asthma, enuresis and stammering the results are good if tension is a dominant factor and can be relieved. Where other factors besides tension are involved, e.g. allergy or dominance, the results are more variable. Here, hypnotherapy may be more effective if combined with other procedures, e.g. skin-testing and desensitization in asthma, and breathing exercises and relaxation in stammering. Much, of course, will depend on the experience and skill of the therapist, the degree of rapport established with the child and his parents, and his clinical ability to treat the child 'as a whole'.

Tension states associated with behaviour-problems or other disorders, are common in children, and their treatment is not easy. Much may be gained by the study and application of hypnotherapy, which by relieving tension, helps the child to adjust itself adequately to its environment. Good results have been obtained, both in private and hospital practice, in a number of children treated along the lines indicated above.

I. Mirvish

90 St. George's Street
Cape Town
18 March 1958

DUCKTAILS

To the Editor: I must first tender my apologies for my rash attempt to invade the pages of a medical journal, being as I am but a layman of little consequence. However, even a layman can diagnose a broken leg is he sees bone protruding from the flesh. My object is only to enlist the support of your many qualified readers.

I suppose there are few who do not know that the inhabitants of Father Flanagan's Boy's Town were, among other types, criminals, gangsters and perverts of both sexes, and of all religions or of none.

The ducktail in our midst is generally, I believe it fair to state, a person with a warped or a weak mind and appears to need to form gangs of which one or two stronger minds act as leaders, for the weak mind ever seeks a stronger one on which to lean and from which to draw inspiration. I further venture to suggest that the great majority of ducktails are what they are because of some maladjusted parent-child relationship; they have, I believe grown up without that sense of security which is essential to every child from the moment of its birth.

Some little while ago, there was a suggestion that Burnside, a village in Northern Natal which became deserted when the colliery over which it stood ceased production, should be converted into a 'Boy (and of course, Girls') Town, but the idea was abandoned because it was felt that it would prove too costly. I venture to state that the abandoning of the scheme will eventually prove (if it has not already proved) far more costly—the cost of unnecessary habitual criminals who could have become decent citizens—a drain on, instead of a source of accrual to, public funds.

I believe that if some of the great names in medicine in South Africa could combine with leaders of religious and social organizations and with magistrates and police, ways and means could be found to set up a Boys' and Girls' Town in South Africa of which the ex-inhabitants would bear no stigma when deemed fit to be sent on to a new life.

If I mention two religious orders among whom leaders of the Towns might be found it is because they were founded, the one (Salesians) by Sr. John Bosco for boys and the other (Good Shepard Sisters) by St. Mary Euphrasia for girls, in both cases for all who were needing and not receiving parental care, but also for the prototype of today's ducktails. However, as was the case with Father Flanagan, the aim of both these orders is not to make bad Jews or bad Methodists and Anglicans into equally bad Catholics, but to make them good Jews, good Methodists and good Anglicans.

One last word about the cost: for a particular purpose the Catholic hierarchy in South Africa set out to collect half a million, then later, one million pounds in 2 years. £750,000 (it looks better in figures) has been collected to date. If the leaders of South African society could be persuaded through the leaders of the medical profession to launch a similar drive, I am certain that the money would be forthcoming for Boy's Town.

Should you find it possible to publish this appeal, Sir, you are welcome to disclose my name and address to any enquirer, but at this stage, I feel it proper to sign myself.

Patrick

14 March 1958

LICENSING OF X-RAY MACHINES

To the Editor: For some 25 years or more, I have urged a scheme for licensing X-ray machines on the Swedish system to protect the public from being exposed to excessive radiation at the hands of those who have not been trained to use X-ray machines.

My advocacy of the Swedish system has apparently not found favour in official quarters but, if ever justification were required, the enclosed anonymous circulars to general practitioners advising them to buy their own X-ray apparatus provides this justification.

One can only assume that this anonymous circular was sent out by a firm which is not amongst the established distributors of X-ray apparatus in South Africa.

Surely licensing of X-ray machines is the only answer to appaling circulars of this type.

M. Weinbren

3-5 Dunkeld Chambers
Smal Street
Johannesburg
18 March 1958

[The circulars enclosed by our correspondent are two anonymous documents entitled 'first' and 'second' 'advice to the medical profession'. The urge that every general practitioner should equip himself with an X-ray apparatus and should X-ray his own patients and send only selected ones to the specialist radiologist. The second circular states: 'When you see a patient for the first time it is absolutely necessary for you, at the end of your examination, to screen his chest. . . . Sometimes you may need to screen a baby. . . . Then comes the filming. . . . Proceeding with your experience and knowledge in radiology, you will perhaps be able to do a barium meal or a barium enema or a cholangiogram or a pyelogram. . . .'. The second circular suggests the voltage and amperage of the machine most suitable for the practitioner, but does not mention any particular machine or firm—*Editor*]